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AN EXPERIMENTAL STUDY OF THE FATE OF THE REMAINING LUNG FOLLOWING TOTAL PNEUMONECTOMY

J. J. LONGACRE, M.D., PH.D., AND RALPH JOHANSMANN, M.D.
(BY INVITATION)
CINCINNATI, OHIO

HISTORICAL REVIEW

A. Anatomic.—Hyperplastic regeneration of the remaining lung following total ablation of one lung in young developing animals has long been recognized (Haasler,[1] 1892), and in a few instances these changes have been contrasted with the dilatation of the definitive lobules occurring in adult mammals (Mohlgaard and Rovsing,[2] 1910; Kawamura,[3] 1914; Knott,[4] 1934). More recently, Hilber[5] (1934) noted in rats that following the removal of one lung there was, at first, increase in size of the remaining lung, due to simple distention of the alveoli. He found, however, that this was gradually replaced by true regeneration of normal lung tissue. In interpreting Hilber's results, it is necessary to consider the fact that in rats the growth factor is present through a considerable part of the life span (Donaldson,[6] 1924).

Rienhoff, Reichert, and Heuer[7] (1935) concluded that the compensatory changes in an adult dog living six months after pneumonectomy consist of a "simple dilatation of the respiratory lobules, made up of the bronchiolus respiratorius, the ductus alveolaris, the atria, the saeculi alveolaris and the alveoli. This dilatation is in no sense an emphysema and there is no interruption or diminution of elastic tissue or fusion of alveoli to suggest pathologic changes in the lung parenchyma."

Bremer,[8] in 1937, clarified the situation by accounting for the disparity in the above observations. He definitely demonstrated: (1) that following removal of one lung in a kitten, actual regeneration of new alveoli

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From the University of Cincinnati College of Medicine, Departments of Surgery and Pathology, Cincinnati General Hospital, and the Institute of Medical Research, Christ Hospital.

and respiratory units occurred, marked by the presence of tubular sprouts, indicative of normal growth; (2) that following ablation of one lung in a mature cat, enlargement of the remaining lung is brought about by the dilatation of the alveoli and distention of respiratory units. These observations were made one month after pneumonectomy.

Reinhoff[9] (1936) claimed that there was no evidence to indicate that compensatory dilatation of the lung was harmful. Graham,[10] in the discussion of Reinhoff's paper, raised the question: "I am not convinced that as the years go by such patients may not suffer from the effects of emphysema, which may be serious in themselves. It is possible, therefore, that perhaps five to ten years from now all of us may feel that a partial or complete thoracoplasty should be performed in many of these cases, although not necessarily at the time of, or even soon after, the operation for the removal of the lung."

B. Physiologic.—From the time of Biondi's[11] early experiments (1884) it has been known that animals could survive the removal of one lung. It was not, however, until the work of Heuer and Andrus[12] (1922) that any attempt was made to study the compensatory readjustments of the organism to pneumonectomy. With their animals under resting conditions, they noted following pneumonectomy:

1. An increase in alveolar carbon dioxide, lasting for thirty days.
2. A fall in alveolar oxygen, returning to the preoperative level in from twenty-eight to sixty-six days.
3. A slight increase in the blood carbon dioxide, falling to normal by the twenty-fifth day.
4. A marked fall in the blood oxygen content up to the eleventh day, returning to normal by the twenty-fifth to thirtieth day.

In 1923 Andrus[13] reported a decrease of 42 per cent in the total lung volume immediately following pneumonectomy, with return to the preoperative level in from twenty to thirty-six days. Drastich, Adams, Hastings, and Compére[14] (1934) reported that reduction in active pulmonary tissue by 50 per cent does not embarrass the organism when engaged in moderate exercise (running on the level for thirty minutes), but that more strenuous exercise results in the formation of more fixed acid than normal.

Longacre, Carter, and Quill[15] (1937), in a series of "strain experiments" designed to assay the efficiency of the cardiorespiratory system after pneumonectomy, under conditions of increased oxygen demand by the tissues, noted the following facts: Immediately after pneumonectomy the cardiorespiratory reserve is cut in half as the result of the removal of approximately 50 per cent of the functional diffusing lung surface area. This is sufficient for resting conditions and moderate exercise. But as the amount of strain is increased, the degree of impairment of the cardiorespiratory reserve becomes increasingly apparent. On succeeding monthly runs there is a tendency for the animals to show less

and less embarrassment, due to compensatory changes (increase in tidal air, gradually increasing subtidal lung volume of the remaining lung, a steady increase in the hemoglobin). However, as to dogs subjected to pneumonectomy after they had attained their maturity, there is still impairment of the cardiorespiratory reserve even six to nine months postpneumonectomy, as indicated by their failure to saturate their arterial blood as efficiently as before pneumonectomy and by their inability to eliminate heat during a two-hour run on the treadmill. Also, in terms of absolute strain (anoxemia test), this same impairment is evident, indicating that the animals have come back only 75 to 80 per cent of the way. However, in animals having total pneumonectomy as puppies, no impairment was noted when they were compared with their normal control mates (Carter, Longacre, Quill,[16] 1938). These results indicate that the capacity of the organism for further growth accounts not only for the difference in the anatomic changes, but also for the difference in the degree of return of function.

EXPERIMENTAL STUDY

Thus far, the studies of the compensatory readjustments following pneumonectomy have covered but a relatively short period. The following problems still remain to be solved: 1. What is the fate of the remaining lung after years of carrying the added strain? 2. Will this fate be identical in those young developing animals operated upon while the growth factor is still present as compared with animals subjected to pneumonectomy after maturity is reached? 3. In time, will the compensatory dilatation noted in the adult animals following pneumonectomy terminate in true pathologic emphysema with all of its embarrassing effects?

To this end, a group of dogs (some operated upon as puppies, others having pneumonectomy as adult animals) have been followed, studied, and sacrificed at various intervals up to four years, actually about one-half of their normal life expectancy.

Determination of the intrapleural pressure may be used as an index of the integrity of the elasticity of the lung. If the intrapleural pressures remain constant, it may be assumed that the elastic tissue in the lung is not giving away under the increased strain, but if it approaches atmospheric pressure, it will indicate that the elastic tissue is breaking up, resulting in emphysema.

The intrapleural pressure in a series of normal dogs had a range of from –5 to –9 cm. of water, with an average variation of 4 cm. of water pressure between the height of a normal expiration and inspiration. In two animals, one subjected to pneumonectomy as a puppy and one as an adult, the intrapleural pressure at the end of four years was found to be lower and to have an average variation of only 3 cm. of water pressure (puppy, –3.5 to –6.5 cm.; adult, –4 to –7 cm.). The normal control mate of the same litter, at the end of the four-year period, still had a normal

range of –5 to –9 cm. of water. Thus, in both the animals there seemed to be a loss of elastic recoil, as compared to their control mates.

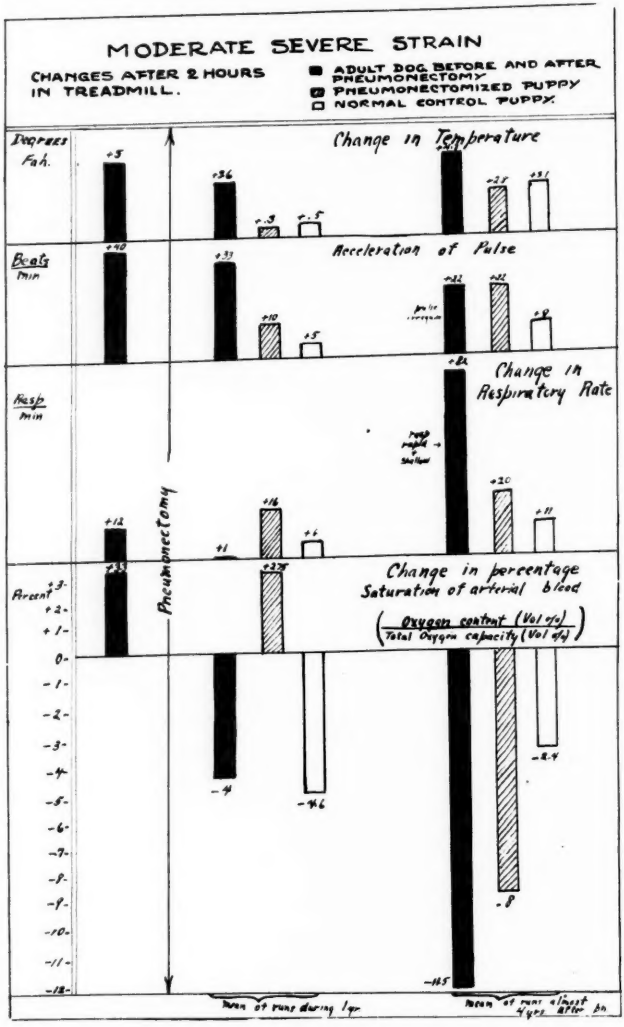


Fig. 1.

Moderate Severe Strain.—The strain experiments were repeated almost four years following pneumonectomy on the three animals which had not been sacrificed and which had survived the vicissitudes of the "doghouse." One of these was a dog subjected to pneumonectomy as a puppy, another its control mate, and the other an adult dog which was operated upon during the same period.

TABLE I

TWO HOURS IN TREADMILL

	TEMPERATURE (° F.)		PULSE		RESPIRATION		ARTERIAL BLOOD					HEMOGLOBIN (GM.)	ANOXEMIA TEST (%)
							O_2 CONTENT		O_2 CAPACITY		CHANGE IN O_2 SATURATION (%)		
	BEFORE EXERCISE	AFTER EXERCISE	BEFORE EXERCISE	AFTER EXERCISE	BEFORE EXERCISE	AFTER EXERCISE	BEFORE EXERCISE	AFTER EXERCISE	BEFORE EXERCISE	AFTER EXERCISE			
Adult Series													
No. 619													
Before pneumonectomy	101.0	106.0	100	140	24	34	24.9	27.4	27.2	28.9	+3.3	20.02	2.2
6 mo. after pneumonectomy	101.4	106.0	104	150	20	22	23.2	22.7	24.6	25.0	−3.5	18.35	2.5
9 mo. after pneumonectomy	101.4	103.8	110	130	20	20	25.5	23.2	27.5	26.3	−4.5	20.5	2.4
3 yr. and 9 mo. after pneumonectomy	101.4	107.6	100	110 (irregular)	20	160 (shallow)	23.36	18.85	23.75	21.8	−12.0	17.7	−4.96
4 yr. after pneumonectomy	101.0	104.4	128	160	20	44	17.83	15.88	20.02	20.4	−11.0	15.07	
Puppy Series													
No. 625, Puppy subjected to pneumonectomy													
6 mo. after pneumonectomy	103.0	103.4	100	120	20	30	18.9	21.1	19.0	20.6	+2.6	14.1	3.7
9 mo. after pneumonectomy	102.0	102.2	120	120	20	26	20.6	19.5	22.8	20.9	+2.9	18.5	2.8
3 yr. and 9 mo. after pneumonectomy	102.4	105.0	96	100	16	32	19.23	16.4	21.05	20.07	−9.0	15.0	−3.32
4 yr. after pneumonectomy	101.4	104.4	100	140	16	24	19.21	18.15	20.45	20.80	−7.0	15.2	
No. 629, Control puppy													
6 mo. after pneumonectomy													
9 mo. after pneumonectomy	103.0	103.0	80	80	20	20	19.6	16.7	21.2	20.1	−4.4	15.8	5.4
3 yr. and 9 mo. after pneumonectomy	102.5	103.5	90	100	24	36	19.7	18.0	21.5	20.7	−4.6	16.0	−3.8
4 yr. after pneumonectomy	100.8	104.6	80	90	14	32	18.4	19.89	20.4	21.15	+1.8	15.9	−3.1
	101.4	104.0	84	92	16	20	21.10	20.12	22.20	22.95	−6.0	17.1	

Following the two-hour runs, almost four years after pneumonectomy, the control of the puppy series held as good a record as on its runs three years previously. On the other hand, the animal which was operated upon while a puppy showed considerably more embarrassment as far as the degree of elevation of pulse and respiration was concerned. The same degree of embarrassment was noted in its inability to eliminate heat and to saturate the arterial blood. Despite the degree of embarrassment, this animal did not show the complete exhaustion and changes demonstrated by the animal which was subjected to pneumonectomy as an adult. On the observations six to nine months following pneumonectomy, this animal had shown considerable embarrassment, but never the complete and utter exhaustion demonstrated at the present time. On one of the runs the temperature rose from 101.4° to 107.6° F., pulse became irregular, respirations extremely rapid and shallow, and the saturation of the arterial blood fell to an all-time low of –12 per cent after the two-hour run. The runs were repeated for all three animals, with comparable results. (Table I and Fig. 1.)

Absolute Strain as Measured by the Anoxemia Test.—In our earlier work the anoxemia test was developed to put the ultimate degree of strain on the cardiorespiratory unit and still have the animal survive. The animals mere made to breathe room air in a closed system, the carbon dioxide being removed and the oxygen being gradually consumed by the animal to a certain critical level recognized by loss of consciousness, loss of sphincter control, dilatation of the pupil, and cessation of respiration. At this point, although all respirations have ceased, the heart will still be beating. The animals were carried to this endpoint and were then cut out of the system by means of a three-way valve and resuscitated. Samples taken of the respired air in the closed system at the end of the test were collected over mercury in Hempel pipettes and then analyzed in a Haldane apparatus.

The value for the normal puppy control was slightly lower than the level three years previously (3.1% as against 3.8%). This is in agreement with previous observations, that, as the basal metabolic rate falls with the animal reaching maturity, the critical oxygen level reaches its lowest base line. The value for the animal treated by pneumonectomy as a puppy, on the other hand, was higher than its previous record (3.32% as against 2.8%). The critical level for the animal subjected to pneumonectomy as an adult was twice as great as its previous levels (4.96% as against 2.5% nine months after pneumonectomy and 2.2% before pneumonectomy), indicating very definitely that the cardiorespiratory reserve is but one-half of its level of three years previously and less than one-half of its original value before pneumonectomy. (Fig. 2.)

As has been shown in our earlier work, following pneumonectomy the cardiorespiratory reserve is cut in half by the removal of 50 per

cent of the functional diffusing surface area, but within nine to twelve months animals operated upon as adults will come back 75 to 80 per cent of the way. Interpreting the results of the anoxemia test in previous experiments, it would appear that the amount of compensatory return of function occurring during the first year is lost in varying degrees during the next three years. This degree of change is maximal in

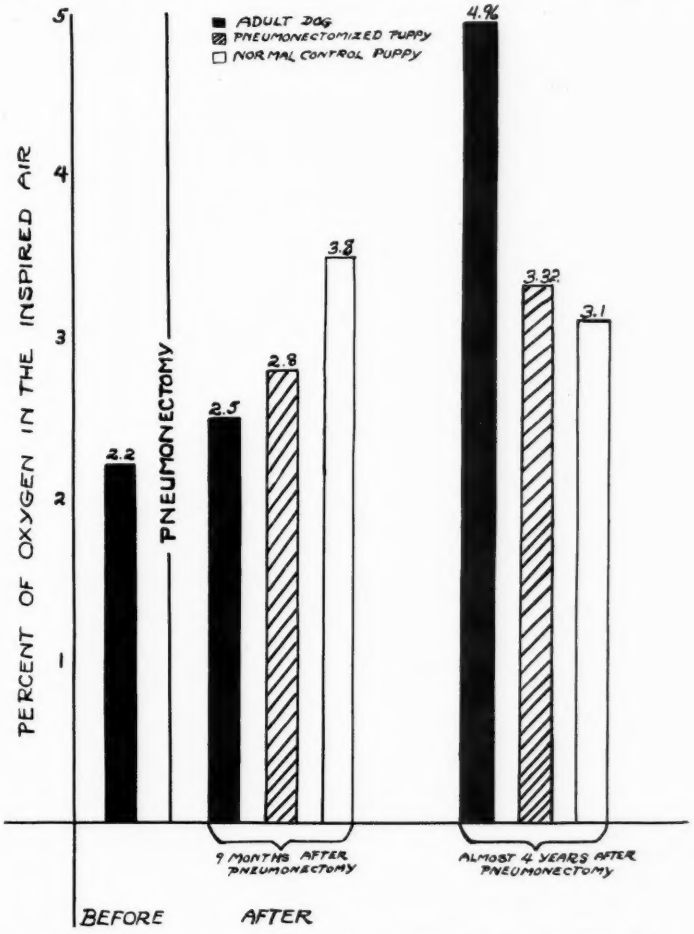


Fig. 2.—Chart showing absolute strain as measured by the anoxemia test.

those animals treated by pneumonectomy as adults and minimal in those animals operated upon while the growth factor is still present. This same retrogressive change is shown by the aforementioned moderate strain experiments. Furthermore, the fall in intrapleural pressure indicates a beginning loss of elastic recoil in the remaining lung.

HISTOLOGIC STUDY

Two normal control dogs, three dogs subjected to pneumonectomy as puppies, and two dogs operated upon as adults were sacrificed by intravenous injection of ether. The three dogs of the puppy series were sacrificed two, three, and almost four years after pneumonectomy. One dog of the adult series was sacrificed two years, and the other almost four

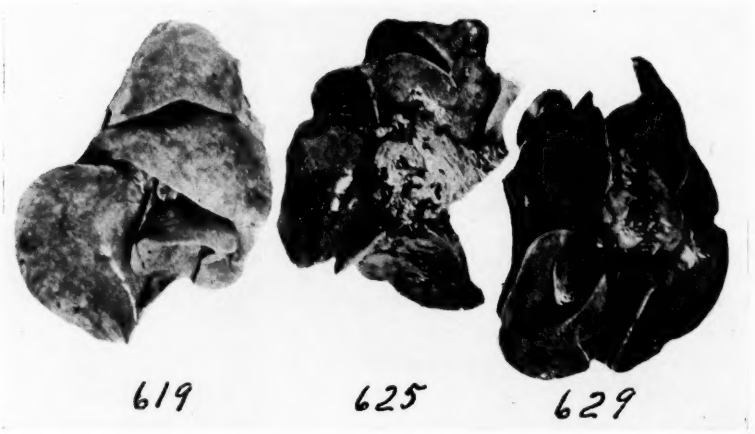


Fig. 3.—Lungs of three dogs after fixation in Bouin's fluid. No. *619*, the remaining lung from a dog operated upon four years previously as a young adult; No. *625*, the remaining lung from a dog subjected to pneumonectomy four years previously as a 1-month-old puppy; No. *629*, the lungs of a normal control dog.

years, after pneumonectomy. On opening the thoracic cage of the dogs in which a lung had been removed, the entire mediastinum was pushed toward the side of the pneumonectomy and the upper two-thirds of the upper lobe of the remaining lung was herniated into the upper portion

TABLE II

	AVERAGE DIAMETER OF ALVEOLI ALONG PLEURAL SURFACE (μ)	AVERAGE DIAMETER OF ALVEOLI ALONG BLOOD VESSELS AND BRONCHI (μ)	AVERAGE (μ)
Adult Series			
Normal control	107	104	106
No. 564—2 yr. postpneumonectomy	128	130	129
No. 619—Almost 4 yr. postpneumonectomy	128	130	129
Puppy Series			
No. 629—Normal control	118	114	116
No. 624—2 yr. postpneumonectomy	91	100	96
No. 626—3 yr. postpneumonectomy	95	91	93
No. 625—Almost 4 yr. postpneumonectomy	101	107	104

of the opposite pleural cavity, while the posterior lower lobe had herniated into the lower portion of the opposite pleural cavity. The lungs of the two normal control dogs and the remaining lung of each of the three

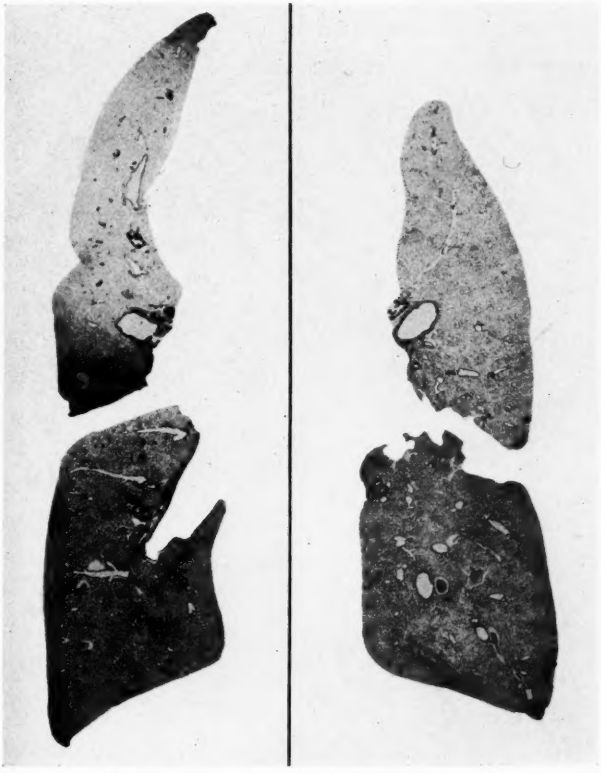


Fig. 4. Fig. 5.

Fig. 4.—Large histologic section of lung of normal control dog.
Fig. 5.—Large histologic section of remaining lung of dog operated upon four years previously as a puppy.

dogs operated upon as puppies and the two adult dogs which had been subjected to pneumonectomy were fixed after the technique suggested by Bremer by intratracheal injection of Bouin's fluid at a constant, low pressure (Fig. 3). Large sections (Figs. 4 to 7) taken for microscopic study from the upper and the posterior lower lobe of each lung represent cross sections of the entire lobes from the hilum to the periphery. An estimate of the average diameter of the alveoli was made by counting the number of alveoli in a field of known diameter. One hundred fields were counted in each lung. The count of alveoli was made of those subjacent to the pleura along the apical, lateral, and diaphragmatic surfaces and of those throughout the deeper portions of the lobes along

the small bronchi and blood vessels which were cut in longitudinal section. The figures obtained from this computation do not necessarily represent the average full diameter of the alveoli but, since all specimens were prepared in a similar manner, the figures may be used for the purposes of comparison. (Table II.)

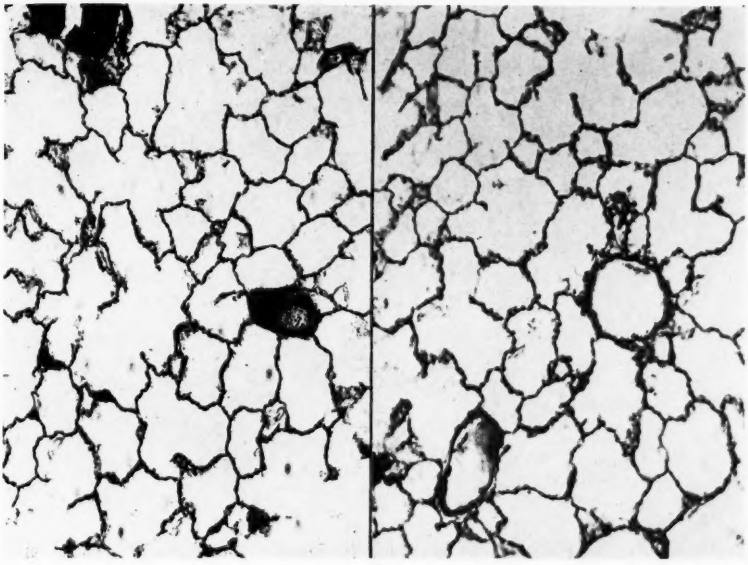
Fig. 6.

Fig. 7.

Fig. 6.—Large histologic section of remaining lung of dog operated upon two years previously as an adult.

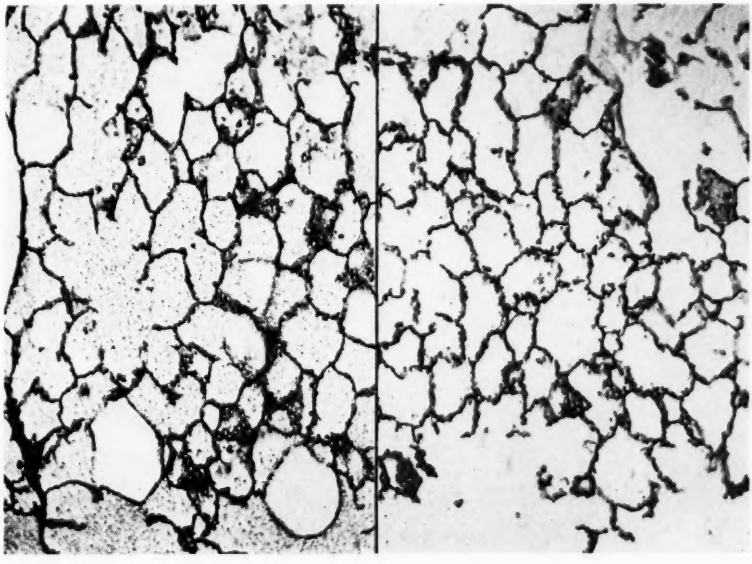
Fig. 7.—Large histologic section of remaining lung of dog subjected to pneumonectomy four years previously as an adult.

As determined by this method, the average diameter of the alveoli of the lungs of the control animals of the puppy and adult series was 116 μ and 106 μ, respectively. A slight decrease in the diameter of the alveoli, 96 μ, 93 μ, and 104 μ, was noted in the remaining lungs of each of the animals operated upon as puppies, which were sacrificed two, three, and almost four years after pneumonectomy. However, there was a slight enlargement of the atria as compared to those of the nor-



A. *B.*

Fig. 8.—*A*, Photomicrograph of peripheral portion of lung of normal control dog. *B*, Hilar portion of lung of normal control dog.



A. *B.*

Fig. 9.—*A*, Photomicrograph of peripheral portion of remaining lung of dog operated upon four years previously as a puppy. Alveoli are approximately the normal size. *B*, Hilar portion of remaining lung of dog treated by pneumonectomy four years previously as a puppy. Alveoli are approximately the normal size.

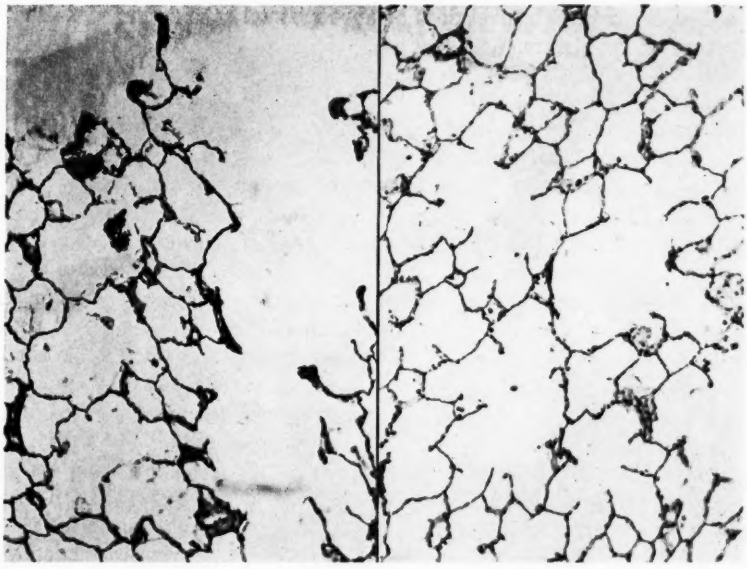
mal control animals. This distention of the atria was evident primarily in the tissue subjacent to the pleura. There were also a few areas in which the walls of the alveoli were broken and in which there was a slight thickening and clubbing of the broken ends. The remaining lung of each of the two dogs subjected to pneumonectomy as adults showed an increase in the average diameter of the alveoli to 129 μ, distention of the atria, and fusion of alveoli due to a breaking of the alveolar walls. This break of the alveolar septa, together with thickening and clubbing of the ruptured ends and collapse of capillaries, was principally evident in the peripheral portions of the lung and was more apparent in the dog sacrificed almost four years after pneumonectomy than in the dog sacrificed two years after pneumonectomy. (Figs. 8 to 11.)

Fig. 10.—Photomicrograph of peripheral portion of remaining lung of dog operated upon two years previously as an adult. Alveoli are distended, and there is some breaking of alveolar septa and slight clubbing of the broken ends.

Duplicate sections were stained with Weigert's elastic tissue stain in an attempt to demonstrate evidence of fragmentation of the elastic tissue fibrils. The elastic tissue was distributed quite evenly throughout the pleura and the alveolar walls in the lungs of the two normal control dogs.

In the three dogs operated upon as puppies there were a few areas in which the elastic tissue appeared slightly fragmented. However, in general, the distribution of elastic tissue appeared much the same as was noted in the control animals.

In the adult dogs the ends of the ruptured alveolar walls showed a clumping of fibrils of elastic tissue in their thickened ends. There appeared, in addition, to be a suggestion of fragmentation of the elastic tissue in many of the alveolar walls. This was more suggestive in the four-year postpneumonectomy dog than in the dog sacrificed two years after pneumonectomy. This evidence of fragmentation must be considered subjective since objective demonstration has been difficult to obtain.



A. *B.*

Fig. 11.—*A*, Photomicrograph of peripheral portion of remaining lung of dog subjected to pneumonectomy four years previously as an adult. Alveoli are distended and there is breaking of alveolar septa, with clubbing of the broken ends. *B*, Hilar portion of remaining lung of dog operated upon four years previously as an adult. Alveoli are distended, and there is breaking of alveolar septa.

The histologic picture of the lungs in both the puppy and adult dog series seems to be correlated with the results of the previous physiologic experiments. The small size of the alveoli in the remaining lung of each of the dogs subjected to pneumonectomy as a puppy indicates hyperplasia of the respiratory units since each of these lungs was almost twice the size of a normal lung. However, there is a very slight tendency toward dilatation of the atria and fusion of alveoli three to four years after pneumonectomy. The dogs of this series withstood the moderate and severe strain experiments very well and showed only a small decrease

of lung function three to four years after pneumonectomy as compared to their nearly perfect record nine to twelve months after pneumonectomy.

There is no evidence histologically of hyperplasia in the remaining lungs of the dogs operated upon as adults, but rather a dilatation of the respiratory units and evidence of the development of chronic emphysema as seen in the breaking of the alveolar walls, thickening and clubbing of their broken ends, collapse of capillaries, and possible fragmentation of elastic tissue. Since these dogs were subjected to pneumonectomy as young adults, the emphysematous changes occurring cannot be ascribed to old age because the animals were sacrificed when only one-half their normal span of life had been completed and also because the normal control dogs of approximately the same age did not show a similar picture.

DISCUSSION AND SUMMARY

According to Eppinger and Schauenstein[17] (1904), and Orsos[18] (1907), the functional impairment of the elastic tissue fibers in the lungs of asthmatic persons is secondary to the overstretching of the alveoli. More recently, Christie[19] demonstrated that the functional impairment is definitely correlated with the loss of elastic recoil of the lungs. Also, with the loss of elasticity the peripheral alveoli which are distended and relatively ischemic are overventilated at the expense of the deeper and more normal alveoli. Therefore, the ventilation of the more peripheral portions must be largely wasted effort since they contain a much diminished portion of the pulmonary circulation. Such a change would be reflected in the decrease of the effective tidal air as some of the air is shunted into ineffective overdistended alveoli deficient in blood supply. As respiratory compensation fails, insufficient aeration of the blood occurs and a vicious cycle is established, with increasing demand for ventilation and decreasing ability to ventilate.

Miller and Rappaport[20] (1938) clearly define the intrinsic function of the lung as "the creation, maintenance and regulation of internal surface adequate in quantity and quality for momentary requirements of gas exchange and fluid elimination under all conditions." The functional diffusing surface area of the lung of the puppies was decreased approximately 50% immediately following pneumonectomy. During the nine to twelve months after pneumonectomy, creation of adequate diffusing surface apparently had been achieved by hyperplasia and regeneration of the respiratory units and this had been maintained fairly adequately in the succeeding years. The failure of these dogs to maintain nearly perfect records three and almost four years after pneumonectomy is possibly due to a slight decrease of this functional diffusing surface area as evidenced by beginning fusion of some of the alveoli.

In the adult dogs treated by pneumonectomy this internal lung surface apparently had never been adequate. Although expansion of the re-

spiratory units did occur with the return of the subtidal lung volume to practically the normal level, there was only approximately 75 to 80% restoration of function nine to twelve months after pneumonectomy. This increase of air space without simultaneous creation of corresponding functional diffusing surface constitutes a compensatory emphysema. However, after this initial dilatation of the respiratory unit, there is a breaking of the alveolar walls, collapse of the capillaries, and, as evidenced by an increase of the intrathoracic pressure, a diminution of elastic recoil, with the onset as shown histologically of a true chronic interstitial emphysema resulting in decrease of the functional diffusing surface area of the lung, decreased capacity to oxygenate the arterial blood and to eliminate heat, and, consequently, increased inability of these adult dogs subjected to pneumonectomy to withstand severe strain.

These experiments present evidence that no alteration of the procedure of pneumonectomy is indicated at the present time during the period in which the growth factor is still present, as it appears that the hyperplasia and regeneration of the remaining lung is sufficient to produce a functional diffusing surface area which is adequate in quantity and quality for gas exchange and fluid elimination under practically all conditions of strain. However, as the dogs of the puppy series of this experiment were operated upon at 1 month of age, more experimental work is indicated to determine any variation in degree of regeneration and restoration of function of the remaining lung during different segments of the "curve of motion of growth."

The results of the physiologic experiments performed on the dogs operated upon as adults picture, as predicted several years ago by Graham, the decrease of function of the remaining lung four years after pneumonectomy. These results are confirmed histologically by the demonstration of the onset of chronic interstitial emphysema.

If this overstretching of the remaining lung over an extended period of time, with its subsequent diminution of elasticity, could be prevented, the later development of chronic interstitial emphysema might be averted. However, no direct evidence can be gleaned from these present experiments that either partial or complete thoracoplasty performed either at the time of or following pneumonectomy will prevent overstretching of the respiratory units, with the subsequent embarrassing sequelae. Another series of experiments is to be undertaken in which young mature animals are to be subjected to pneumonectomy, some of them to be subsequently subjected to thoracoplasty and others kept as controls. These animals are to be assayed in terms of response to severe and absolute strain over a period of years.

We are indebted to Dr. R. S. Austin for his assistance in evaluating the degree of pathologic change, to the services of the Cardiac Laboratory under the supervision of Dr. Johnson McGuire for blood-gas and gas analyses, and to Dr. James N. Christiansen, Director of the Pneumothorax Clinic, for his determination of intrapleural pressures.

REFERENCES

1. Haasler, F.: Virchows Arch. f. path. Anat. **128**: 527, 1892.
2. Möhlgaard, H., and Rovsing, T.: Zentralbl f. Chir. **37**: 1276, 1910.
3. Kawamura, K.: Deutsche Ztschr. f. Chir., Leipzig, 1914.
4. Knott, A.: Ztschr. f. Kinderh. **56**: 338, 1934.
5. Hilber, H.: Morphol. Jahrb. **74**: 171, 1934.
6. Donaldson, H. H.: The Rat, Memoirs of the Wistar Institute, ed. 2, no. 6, Philadelphia, 1924, Wistar Institute of Anatomy and Biology.
7. Rienhoff, W. F., Jr., Reichert, F. L., and Heuer, G. J.: Bull. Johns Hopkins Hosp. **57**: 373, 1935.
8. Bremer, J. L.: J. THORACIC SURG. **6**: 336, 1937.
9. Rienhoff, W. F., Jr.: South M. J. **29**: 447, 1936.
10. Graham, Evarts A.: Discussion of Rienhoff's paper, South. M. J. **29**: 447, 1936.
11. Biondi: Wien. med. Jahrb. **1**: 207, 1884.
12. Heuer, G. J., and Andrus, W. D. D.: Bull. Johns Hopkins Hosp. **33**: 130, 1922.
13. Andrus, W. D. W.: Bull. Johns Hopkins Hosp. **34**: 119, 1923.
14. Drastich, L., Adams, W. E., Hastings, A. B., and Compére, C. L.: J. THORACIC SURG. **3**: 340, 1933.
15. Longacre, J. J., Carter, B. N., and Quill, L. McG.: J. THORACIC SURG. **6**: 237, 1937.
16. Carter, B. N., Longacre, J. J., and Quill, L. McG.: J. THORACIC SURG. **7**: 329, 1938.
17. Eppinger, H., and Schauenstein, W.: Ergebn. d. allg. Path. u. path. Anat. **8**: 267, 1904.
18. Orsos, F.: Beitr. z. path. Anat. u. z. allg. Path. **41**: 95, 1907.
19. Christie, R. V.: J. Clin. Investigation **13**: 395, 1934.
20. Miller, J. A., and Rappaport, I.: Ann. Int. Med. **11**: 1644, 1938.

DISCUSSION

DR. CHARLES W. LESTER, New York.—Dr. Longacre is to be congratulated on a very excellent piece of research. His figures will have a good deal of bearing on the treatment of cases of pneumonectomy, in the future, I am sure. It is interesting to note that his first impressions, derived three years ago when he studied these dogs at the end of that period, have changed somewhat as the dogs have grown older, and it rather bears out the contention that has been made a good many times, that increasing the size of the alveolus does not necessarily increase its function, but in time certainly tends to decrease it.

We have not done any laboratory work, such as Dr. Longacre has done, but we have made some functional studies on patients after pneumonectomy. I would like to give you briefly the results of some of these studies. Of course, we have not been able to control them with the careful control that Dr. Longacre has been able to give his dogs in the laboratory, and in addition, these patients are all suffering from a very extensive pulmonary disease, which also has a bearing on the result.

Three patients have been studied during the last year, who have undergone pneumonectomy by Dr. Berry for carcinoma, and they have been studied by Dr. Cournand, who will report them later.

The first patient is a man, 60 years old, who, in addition to carcinoma of the left lung which was completely collapsed, had auricular fibrillation. His left lung was removed and he had a rather stormy course, but six months after that his vital capacity was about one-half what would be expected for a man of his age with both lungs. The residual air was about 34%, which indicates a certain amount of emphysema. However, he could saturate his arterial blood with oxygen by breathing 6 L. of air a minute.

Under exercise, and the exercise in these cases consists in stepping up and down a step thirty times, he was still able to saturate his arterial blood, but the ventilation had to increase so much that he was ventilating at the rate of about 34 L. per minute,

In other words, this man had a fairly good lung for some purposes, but it was by no means a functionally perfect lung.

The next man was a younger man of 31 years, who also had a carcinoma of the left lung which had completely collapsed that lung. Prior to operation, he had only one-half the estimated vital capacity, but he had a fairly good functioning lung. The first month after operation the figures dropped considerably. Four months later, and this is the astonishing point, the oxygen saturation of his arterial blood was higher than it had been before operation, the gaseous exchange in his alveoli was better and more effective, and the pulmonary function was really improved by operation.

Now, whether or not this is going to continue remains to be seen. From Dr. Longacre's figures, I am tempted to advise thoracoplasty, so that this man will continue to hold the gain which he has already maintained and not lose it because of emphysema.

The third case differs materially from Dr. Longacre's figures and shows that the pathologic condition of the lung can change the situation materially. The patient, on first consideration, was the most hopeful of all the cases. He had a small bronchial carcinoma in the right middle lobe, and the upper and lower lobes on that side were fairly well aerated. Figures before operation were fairly close to normal, except that the patient had a little emphysema. At operation, it was found that there was already metastasis in the mediastinum.

One month after operation the figures had all dropped materially, vital capacity was less than one-half, and he could not saturate his arterial blood at rest. On exercise, the oxygen content of his arterial blood was even lower. He was not getting a normal gaseous interchange in the alveoli. Four months after operation, this bad condition was even worse. Eight months after operation, he died of metastases, and at autopsy a strange feature was discovered. Although the right chest was filled with various things, including thickened pleura and metastatic tumor, his left lung had not pushed over there at all. He had a little microscopic emphysema, but he had died with the pulmonary function greatly decreased not because of operation, but because of disease.

We have also had an opportunity to follow several children upon whom pneumonectomy was performed by Dr. Bohrer and myself, for periods of from ten months to two and one-half years. These children have done very well. We are not able to give them tests to check up on this, but they are able to carry on their play and behave clinically as other children of their age.

DR. EVARTS A. GRAHAM, St. Louis.—I think we all are greatly indebted to Dr. Longacre for this magnificent, painstaking piece of work, extending over a long period of years. It is the most definite information which we could possibly have on this much discussed question as to what happens to the other lung after a total pneumonectomy.

I am tremendously interested, of course, in the fact that my original fears of what would happen to the other lung seem to be confirmed by this magnificent bit of experimental work.

In the first case of total pneumonectomy for carcinoma, the large pleural space that was left seemed to be of such enormous size that it was overwhelming. It seemed to me at the time that obviously the best thing would be to obliterate this space, and consequently seven ribs were removed at the same time that the lung was removed. These were the lower seven ribs. The purpose was to obliterate the pleural cavity as much as possible. Since that time there has, of course, been a good deal of controversy as to whether or not thoracoplasty is desirable or necessary in these cases.

I must say that with the work of Rienhoff, particularly, and others, I became rather convinced that thoracoplasty would seldom be necessary, perhaps never neces-

sary, as a preliminary procedure in these cases. I am more tempted now to think, however, that perhaps it may be necessary, much more often than one might have thought two or three years ago.

In regard to this first patient, who has gone the longest time, now in his eighth year, following a total pneumonectomy for carcinoma, he had a thoracoplasty, as I have just said. He is now 57 years of age. He has no more apparent dyspnea on exertion than the average man of his age. He has, as a matter of fact, far less dyspnea on exertion than I have, as tested on numerous occasions. I do not know of a recent vital capacity determination on him, but in view of Dr. Longacre's work, I cannot help thinking that perhaps the thoracoplasty that was done at the time of the pneumonectomy has saved him from an emphysema which, during the more than seven years which have gone by since the operation, he might have developed to give him serious symptoms.

Now, besides the mere questions of what happens to the other lung and whether or not emphysema develops, there is another feature of this problem which I think has not been emphasized sufficiently, which may be a source of dyspnea and unpleasant symptoms to the patient. I refer to a kinking of the large vessels in the mediastinum by a considerable shifting to one side.

I have had one case of a young negro boy, about 10 years of age, who had a failure of development of one lung, with the result that, as he grew, his trachea and great vessels were pulled clear over to the opposite side. This boy was unable to play as other boys of his age could do, and it was for that reason that his mother brought him around to see what might be done; it was then that it was discovered that he had this mechanical defect. A thoracoplasty promptly relieved him of all his symptoms because it permitted straightening of the trachea and the great vessels. Now this boy, on whom a thoracoplasty was done about three years ago, is able to do everything that any normal boy can do.

Of course, everybody also has had numerous experiences in cases of marked atelectasis and tuberculosis in which the trachea and great vessels have been drawn over to the affected side. Many surgeons have had the experience of seeing these patients immediately relieved of their symptoms of dyspnea merely by a thoracoplasty to straighten out the kinked great vessels, which I think is a more important consideration than a kinking of the trachea, of which I doubt the existence.

There is no use of my going on with these considerations. Time is rapidly passing, but I do want to express my very great admiration for this careful, painstaking work of Dr. Longacre and his associates. I think it is a great contribution to thoracic surgery.

DR. WILLIAM E. ADAMS, Chicago.—I have been interested in this subject for several years, and I wish to say that our results, where the experiments were carried out similarly to those of Dr. Longacre, concur with his in every respect. The frequent clinical need for the removal of a considerable portion of the lung, not only from one side but from both sides, led us to investigate the effect on dogs of bilateral resection of lung tissue, and in some cases the lung function was further reduced by stenosis of bronchi to collapse most of the remaining lung tissue. In some animals the remaining pleural space was partially or completely obliterated by thoracoplasty or by diaphragmatic paralysis, to determine its influence both on the postoperative course and on the structure of the remaining functioning lung tissue.

Our experiments have not been concluded at the present time, only in so far as the physiologic effects of the procedures are concerned. We have some microscopic sections obtained from previous experiments that, as I said, show very much the same changes as those seen in Dr. Longacre's sections.

(Slide) The bronchogram on the left was taken following pneumonectomy on the left side and complete stenosis of the right lower and accessory low bronchi, leaving the right upper and the middle lung lobes functioning. The bronchogram on the right side was made after stenosis of the right middle lobe bronchus; thus only the right upper lobe was functioning. In other words, in this dog was produced what we termed sublethal reduction of lung function.

(Slide) This is a microscopic section through the remaining upper lobe which is atelectatic. You see here, especially along the periphery, but also generally, a marked dilatation of the alveoli, with changes very similar to those present in Dr. Longacre's sections, differing only in degree of severity.

Since we have not sacrificed our animals, I do not know how severe the changes will be in the functioning lung tissue of those dogs in which the diaphragms have been paralyzed or in which a thoracoplasty has been done on one side. It would be logical to suppose that the microscopic appearance would not be altered as much, inasmuch as the remaining functioning lung would not need to hyperinflate to the same degree.

It is interesting to know that a dog with only one lung lobe, a right upper, which represents only approximately 15 per cent of the entire lung, can get along with both diaphragms paralyzed or with one chest entirely collapsed.

Another test to which these animals were put was the ability to tolerate rarified atmospheres. An exhaust chamber was used, the pressure being reduced to assimilate ascension in an airplane as brought out in Armstrong's book on aviation medicine. A normal adult will become unconscious at about 25,000 feet, if the rate of ascension is 5,000 feet a minute. Normal dogs behave in about the same manner. It was interesting to see these dogs with only one lung lobe functioning and with one or both diaphragms paralyzed go up to a similar height before becoming unconscious. About sixteen dogs were tested, with similar results in each animal.

I certainly wish to congratulate Dr. Longacre on a fine piece of well-controlled work.

DR. LONGACRE.—In closing the discussion, I wish to add one other piece of work of which I have just learned from Dr. Payne of Dr. Wangensteen's clinic in Minneapolis. By plicating the diaphragm and so decreasing the excursion of it, and as a result increasing the capacity of the chest, he has been able to produce in practically all instances true changes of emphysema, such as have been shown here by both Dr. Adams and myself.

Time alone, however, will tell with further work whether collapse therapy will prevent this from happening. We believe it will, but we are not certain.

STUDIES IN THE EXPERIMENTAL PRODUCTION OF PULMONARY EMPHYSEMA

JOHN R. PAINE, M.D.
MINNEAPOLIS, MINN.

INTRODUCTION

PULMONARY emphysema has remained an enigma as regards both its etiology and treatment since it was first recognized and described by Laënnec in 1819. In 1861 Rokitansky described the microscopic histology of emphysematous lungs and emphasized the features which are considered typical today: namely, dilatation of the pulmonary alveoli, obliteration of capillaries, fragmentation of the elastic fibers in the alveolar septa, and rupture of the alveolar walls. Theoretically such a picture might be produced by some primary degenerative process of the lung parenchyma. Convincing evidence of any such process, however, is lacking, and at the present time few clinicians believe in its existence. The many and varied theories which have been proposed in the past to explain the etiology of pulmonary emphysema fully attest the uncertainty in our knowledge of the subject. The enumeration of these various theories of etiology would serve no useful purpose. Suffice it to say that at the present time those theories of etiology which merit consideration can be classified under two general heads: (1) some type of obstruction to respiration; and (2) some change in the form or function of the walls of the thoracic cavity. It can be further stated that the majority of writers and investigators believe that some obstruction to the expiratory component of respiration is the basic etiological factor in the production of true pulmonary emphysema in man.

Emphysema produced by obstruction of the expiratory component or of both the expiratory and inspiratory components of respiration can be understood, although in the writings of many of the proponents of these methods the physiologic means by which they operate is not clearly explained. It seems highly improbable that any effective disrupting force can be exerted on the alveolar walls during the course of an expiration, free or forced, because the forces exerted in this phase of respiration act in a centripetal direction; i.e., the intrinsic retraction of the lung and the passive or active collapse of the thoracic walls from the inspiratory to the expiratory position. It can be reasoned, however, that since inspiration is a forceful active process and expiration a

From the Department of Surgery, the University of Minnesota Medical School, Minneapolis, Minn.

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weaker and usually passive process, a continuous distention of the pulmonary alveoli occurs due to the imprisonment of air introduced during inspiration in the presence of obstruction of the trachea or bronchi. A progressive increase in the size of the lung thus occurs until the disrupting forces acting during inspiration finally produce the characteristic fragmentation of elastic fibers and rupture of the alveolar walls. Such a view does not exclude the possibility of disturbances of the circulation in the pulmonary capillaries being a secondary factor. The overdistention of the lung and increased intra-alveolar pressure during expiration in this manner may well produce secondary nutritive disturbances in the lung parenchyma which hasten the rupture of the alveolar walls.

If forces acting in inspiration are considered to be those responsible for the rupture of alveolar walls in the presence of expiratory obstruction, it seems reasonable to assume that the same result might occur in instances where inspiration is obstructed and expiration is free. Under these circumstances it is known that the depth of inspiration and the size of the thorax is greatly increased while the intrapleural pressure becomes much more negative. The stress borne by the lung tissue is therefore proportionally greater. Clinical or experimental proof that emphysema is produced in this way, however, is lacking.

The method by which emphysema can be produced by changes in the form and function of the walls of the thoracic cavity is more difficult to understand. In the past, two types of changes have been thought to be active: namely, (1) those degenerative changes in the upper costal cartilages, first described by Freund in 1858, which were alleged to force the thorax into a relatively fixed inspiratory position; (2) those changes in size and shape of the thoracic cage due to the kyphosis of old age or to the kyphosis of disease of the vertebral column, described first by Loeschcke in 1911 and more recently by Kountz and H. L. Alexander in 1933. Each of these pathologic processes is said to enlarge the thoracic cavity and decrease to a certain extent the respiratory excursion of the ribs. After reviewing the literature on the subject, it seems certain that these structural changes without doubt are frequently found associated with a histologic picture in the lung parenchyma which resembles true pulmonary emphysema, but whether or not they are ever the cause remains obscure. If they are the cause, the method by which they operate requires elucidation.

These theories presumed that an enlargement of the thoracic cavity which would produce an overdistention of the lung leads finally to rupture of the alveolar walls. The increasing experience of thoracic surgeons, however, as indicated recently by Rienhoff, would tend to show that the simple enlargement of the thoracic cavity, uncomplicated by other factors, is probably insufficient to produce true emphysema. It has now been fairly generally accepted that the changes occurring in

the remaining portions of lung following lobectomy or pneumonectomy are not a true emphysema, but a general dilatation of the alveolar spaces without rupture of their walls or fragmentation of the elastic fibers—the so-called compensatory emphysema.

Before the nature of pulmonary emphysema can be understood more fully, it must be demonstrated experimentally whether or not the same histologic picture can be produced by different means. If it can be shown that emphysema is the same end result of different pathologic processes, much will be accomplished toward reconciling the varied theories of its production, clarifying its essential nature, and establishing a rational basis for its treatment.

THE EXPERIMENTAL PRODUCTION OF EMPHYSEMA

Many investigators in the past have claimed to have produced pulmonary emphysema experimentally in animals. The results in some instances have been contradictory and much of the work has been uncontrolled. In addition, such a variety of procedures has been performed with allegedly positive results that, with regards to the etiology of emphysema, an increasing amount of confusion rather than clarification has resulted.

Brown-Sequard was the first investigator who claimed to have produced emphysema in dogs and rabbits. In 1871 he observed pulmonary distention and emphysema following electrical stimulation of the vagus nerves as well as the region of the vagus nucleus in the brain stem. On the basis of other experiments performed at a later date, Brown-Sequard concluded that the production of the emphysema had been due to the contraction of the musculature of the bronchi, which forced the air within their lumina into the terminal alveoli. Brown-Sequard's experiments were of an acute nature and his observations were not checked by any study of histological sections.

In 1877 Köhler partially occluded the tracheas of rabbits with ligatures, and after several weeks found on histologic study that the pulmonary alveoli were dilated, the capillaries in their walls were hyperemic, and the epithelial cells were involved in a fatty type of degeneration. Although these pathologic changes are suggestive of emphysema, Köhler made no such claim. His conclusions were that they were due to some interference with the bronchial musculature and obstruction to the expulsion of mucus.

According to Schall, Hirtz in 1878 reported the production of emphysema in rabbits some weeks following the partial ligation of the trachea. The criteria by which the presence of emphysema was detected are not known.

Sudsuki, in 1899, placed ligatures about the tracheas of six rabbits, but none were placed so as to produce any narrowing of the lumen. In three other rabbits the tracheas were ligated in such a manner that

the lumen was considerably narrowed. This second group of animals was sacrificed after forty-six, sixty, and eighty-four days, respectively. The examination of the lungs both grossly and microscopically is said to have shown the typical pathologic picture of pulmonary emphysema. The lungs of the rabbits in which the trachea was not narrowed by the ligature showed, on the other hand, no evidence of emphysema.

Bullara, in 1900, claimed to have produced emphysema in three dogs by obstructing their nares for a period of time varying from twelve to thirty days. The criteria by which emphysema was judged to be present were not well defined.

Schall, in 1909, reported the results of attempts to produce experimental pulmonary emphysema in dogs by the use of a specially constructed mask which his dogs were trained to wear except when eating (about twenty-two hours a day). Valves on this mask could be so regulated that expiration or inspiration could be obstructed at will. In one dog, inspiration was obstructed for nine months; in another, expiration was obstructed for ten and one-half months; and in still another both expiration and inspiration were obstructed for eleven months. In two animals, acute experiments were performed, with inspiration and expiration greatly obstructed, respectively. Both of these animals died a few hours after the masks were applied. Histologic study of the lungs of all these animals was negative for signs of pulmonary emphysema.

In 1917 Friedman and Jackson, while studying the carbon dioxide content of alveolar air, obstructed expiration by means of a valve attached to a T-tube in the trachea in a series of acute experiments. Fraser examined the lungs of these animals and in microscopic sections found definite emphysema to be present.

Harris and Chillingsworth, in 1919, placed ball valves of brass which obstructed expiration in the tracheas of twenty-five dogs. These valves were allowed to remain in place for periods varying from twenty-four hours to three weeks. The gross and microscopic appearance of the lungs of these animals was reported to be typical of pulmonary emphysema. False valves without any mechanism to obstruct one phase of respiration more than the other were placed in some animals for comparable periods of time. The lungs of these animals, while they did not show the extensive changes noted in the group with obstructed expiration, still showed definite emphysematous changes in scattered areas of the lung edges. The authors minimized the significance of these latter changes and stated that such conditions were commonly seen in laboratory dogs killed by a general anesthetic.

In a very significant contribution to the subject, Nissen, in 1927, reported his results in attempts to produce emphysema in dogs by three different methods. (1) The ribs were cut from the sternum in twelve dogs. A metal bar was then fastened to the cut ends of the ribs and forced away from the sternum. In this manner the thoracic cavity was

effectively enlarged, but the procedure itself proved too severe, six of the animals dying within the first twenty-four hours, and the rest being sacrificed within a relatively short time. The lungs of none of these dogs showed evidence of emphysema either macroscopically or microscopically. (2) In three dogs, four rabbits, and three cats, Nissen produced tracheal stenosis and then sacrificed them after three to six months. Eight of these animals appeared to have distended lungs, but emphysema, when present, was confined to small scattered areas. (3) In ten other animals certain of the main bronchi were ligated, and in four additional animals the right or left pulmonary artery was ligated. Both of these procedures produced atelectasis of the involved portion of the lung and necessitated the remaining aerated portions of the lung to distend and to occupy permanently a larger space than before. In both of these groups of animals, emphysema was found consistently in the lungs at sacrifice after several weeks.

Campbell, in 1927, studied the effect of low oxygen tension (60 mm. Hg partial pressure) on cats, monkeys, rabbits, rats, and mice. Emphysema was produced in the lungs of most of the animals, due, Campbell thought, to the hyperpnea.

In 1930, Loeb placed brass plugs 18 mm. in diameter with a hole 4 to 5 mm. in diameter within the tracheas of dogs. These animals were sacrificed at intervals varying from two to fourteen months and the lungs were examined. A few parenchymal changes indicative of emphysema were found, but these were not definite enough to be considered significant.

The results of extensive experimentation were reported in 1932 by Adams and Livingstone. These investigators, after first producing bronchial stenosis and atelectasis by the intrabronchial application of silver nitrate, removed lobes of the lungs in twenty-eight dogs. When these animals were sacrificed from two months to a year later, the remaining pulmonary tissue consistently showed marked emphysema to be present on histologic examination.

Prinzmetal, in 1933, reported that rats kept in tanks in which the pressure was lowered to 350 to 450 mm. Hg for from one to ten weeks developed pulmonary emphysema as shown by histologic study of lung sections.

In 1936, Kountz, H. L. Alexander, and Prinzmetal, using the technique of Adams and Livingstone, removed about 80 per cent of the lung parenchyma in nineteen dogs. They found that after nine to fifty-two months the remaining lung tissue uniformly became emphysematous. These investigators attempted in this way to study the effect of emphysema on the heart.

The most recent investigator, Hinshaw, reported in 1938 the production of pulmonary emphysema in dogs by the insertion of a special ball

valve into the trachea for periods up to eight months. Expiration was obstructed more than inspiration. No details other than a description of the valve were given.

COMMENT

Justifiable criticism can be raised against more than one of the preceding series of experiments, especially with regards to (1) controls, (2) criteria for establishing the presence of emphysema, and (3) the use of acute experiments to produce a chronic pathologic condition. Nevertheless, the evidence which they present seems to indicate that perhaps in the dog, as well as other laboratory animals, certain parenchymal changes in the lungs which resemble the changes found in patients with chronic pulmonary emphysema can be produced by several methods. These methods can be classified as follows:

1. Obstruction of some type to the passage of air through the tracheobronchial tree.
2. Increase in the space which functioning lung tissue is made to occupy.
3. Decrease in the oxygen tension of the air breathed.

A consideration of the pathologic physiology produced by each of these methods shows that one result produced by each and common to all is an increase in the midcapacity of the thorax and consequently an increase in the average stress which must be borne by the alveolar septa of the lungs. Whether or not this thesis is true must be proved by further experiment and study.

METHODS OF INVESTIGATION

Since most of the investigations by which emphysema has been alleged to have been produced in the experimental animal have involved the creation and maintenance of some degree of obstruction to the passage of air through the trachea and bronchial system, it would seem that further attempts to separate and study the relative effect of such obstructions on the two components of the respiratory act, expiration and inspiration, should be made. In addition, it would seem that, if possible, the theories of Freund and Loeschcke should be submitted to experimental trial. Consequently, the investigations here reported are concerned with the study of: effect of respiratory obstruction; and effect of operative procedures tending to enlarge the thoracic cavity.

I. Respiratory Obstruction:

a. Obstruction of Expiration.—To determine the effect of obstructed expiration, specially constructed valves, as shown in Fig. 1, were fastened in the tracheas of ten dogs. These valves, made of brass and plated with silver, consisted of a short tube 15 mm. in diameter within which a thin light door was hinged so that, during expiration, it was prevented from passing beyond the midpoint by a small metal stop. During inspiration, the door was free to open and allow air to pass with no appreciable resistance. A clearance of about 1 mm. between the door and encircling cylinder was left so that some air might pass, even though the valve was closed. At either end of the cylinder, four holes were cut so that the valve could be fastened into the trachea with fine surgical silk.

Insertion of the valves was made under anesthesia produced by the intraperitoneal injection of 35 mg. of sodium pentobarbital per kilogram of body weight. An incision was made in the midline of the neck and the upper portion of the trachea was freed partially from surrounding structures. The trachea was then partially cut across transversely between two cartilaginous rings and the valve was inserted into the lumen. Fine silk sutures threaded through the holes in the cylinder were passed through the wall of the trachea and tied to fix the valve securely in place in such a manner that its hinge lay in the ventral plane of the body at the dorsal side of the trachea. In this position, gravity tended to keep the door of the valve closed and decreased the likelihood of mucus interfering with its operation.

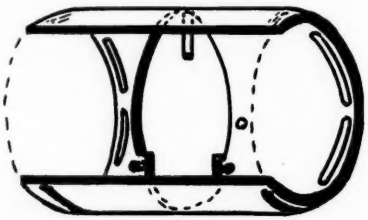

Fig. 1.—Valve inserted into the trachea of dogs to produce respiratory obstruction. Depending upon which way the valve is placed in the trachea, inspiration or expiration is relatively more impeded than the other component of respiration. For control experiments, the hinged door was removed and only the metal cylinder was inserted.

In each dog the intrapleural pressure and the tidal air were determined simultaneously before the insertion of the valve. These measurements were repeated after removal of the valve some weeks or months later when the animal was sacrificed, except in those dogs that died during the experiment. From the measurements thus obtained, the coefficient of pulmonary elasticity according to the method of Christie and McIntosh was determined.* In some instances roentgenograms of the chest were made to determine what effect, if any, the valve would have on the position of the diaphragm. All dogs were examined at frequent intervals to make certain that the valves were functioning. This was easy to ascertain by listening with a stethoscope over the trachea and hearing a distinct click as the hinged door hit against the metal stop.

The animals were sacrificed at varying intervals by the intravenous injection of sodium pentobarbital. The lungs of these dogs, as well as those of others that died during the course of the experiments, were removed from the chest and examined grossly. Several sections were cut from various parts of the lungs, fixed in 1 per cent formalin, and sectioned for histologic study.

b. Obstruction of Inspiration.—The same methods and measurements described above were used to determine the possible effect of obstructed inspiration in the production of emphysematous changes in the lungs of nine dogs by reversing the tracheal valves so that inspiration was impeded and expiration left unobstructed.

c. Control Experiments.—Whereas several investigators have purported to have produced emphysematous changes in the lungs of dogs by the use of certain valves inserted within the trachea and have concluded that the changes produced were due to the effect of obstruction of one of the phases of respiration (usually, expiration), no one to our knowledge has made any control experiments to test this conclusion. The effect of a foreign body which does not appreciably obstruct the passage of

*In another communication the author has indicated that this is the best available method of estimating pulmonary elasticity.

air through the trachea has not been determined experimentally. The valve that has been used in our experiments lends itself well to such control observations.

In three dogs the hinged door of the valve was removed, the tube alone being placed in the trachea and fastened in place with silk sutures. As far as could be determined, the presence of these tubes occasioned the animals no distress and obstructed respiration in no way. As in the other experiments, the elasticity of the lungs was determined before the insertion of the tubes and three months afterward just before the animals were sacrificed by the intravenous injection of sodium pentobarbital. After death, the lungs were inspected grossly and various sections were removed for later histologic study.

II. Effect of Operative Procedures Tending to Enlarge the Thoracic Cavity:

a. Theoretic Basis for the Experiments.—In 1858 Freund called attention to certain degenerative changes which he had noticed in the costal cartilages of patients with pulmonary emphysema. In the following year he published a detailed description of these changes and postulated the theory that certain cases of emphysema were due to a decrease in the elasticity and an increase in the length of the upper costal cartilages. The degenerative process which Freund described consisted of the deposition of calcium within the body of the cartilage and an increase in its length of about 0.5 cm. Such changes, it was reasoned, would increase the size of the thoracic cavity, place the ribs in an inspiratory position, and decrease their elastic recoil. To correct this condition, it was suggested that portions of the upper costal cartilages be excised, thus allowing the ribs to assume a more normal position and to move more freely in respiration.

Little attention was paid to these ideas until 1901, when Freund renewed his investigations. At that time both Kocher and Sauerbruch used Freund's operation and believed in its therapeutic value. After a few years of popularity, however, it fell into disuse, so that at the present time one finds only occasional references to it in the current literature.

Loescheke, in 1911, expressed the opinion that Freund's explanation that rib cartilages in emphysema elongated and thus fixed the ribs in inspiration could not be accepted. He pointed out, on the basis of clinical and post-mortem observations, that in emphysema the entire thorax almost never assumed the inspiratory position; that, on the contrary, some ribs were usually in a relatively expiratory position. His conclusions were that lung distention and emphysema were the consequence of a rigid thorax, but that the primary cause of this was some pathologic process in the spine. This process fixed the spine and produced a kyphosis. The ribs at the level of and above the kyphosis would assume an inspiratory position and those below, an expiratory position. In confirmation of this theory, he was able to find many emphysematous lungs at post-mortem examination in which the severity of emphysematous changes appeared to be determined definitely by the location of the kyphosis.

More recently, Kountz and Alexander have differentiated a nonobstructive type of emphysema from the so-called obstructive type. In these patients a stiffness and straightness of the spine have been noted which have been thought to be due to a degeneration of the intervertebral discs. The result of these processes is said to separate the ribs and produce a barrel-shaped chest. This theory can be justly questioned since the authors have produced no histologic proof that a true emphysema is present in such cases, despite the outward resemblance of the form of the thorax to that known to occur with true pulmonary emphysema. In addition, Kountz and Alexander have admitted that the physiologic responses of patients with nonobstructive emphysema are quite different from those exhibited by the so-called obstructive type.

It is interesting to note that, while these three theories differ as to the exact process by which the emphysema is produced, they all have the one fact in common, that the size of the thoracic cavity is permanently enlarged by some process affecting primarily its walls. The author has attempted to submit these ideas to experimental proof by carrying out certain operative procedures on the dog.

b. Technique of Experiments.—In four dogs an attempt was made to reproduce as closely as possible the conditions which Freund considered to be one of the causes of pulmonary emphysema, namely: (1) decrease in the elasticity of the thoracic wall, and (2) increase in size of the thoracic cavity.

In four other dogs an attempt was made to increase the size of the thoracic cavity only.

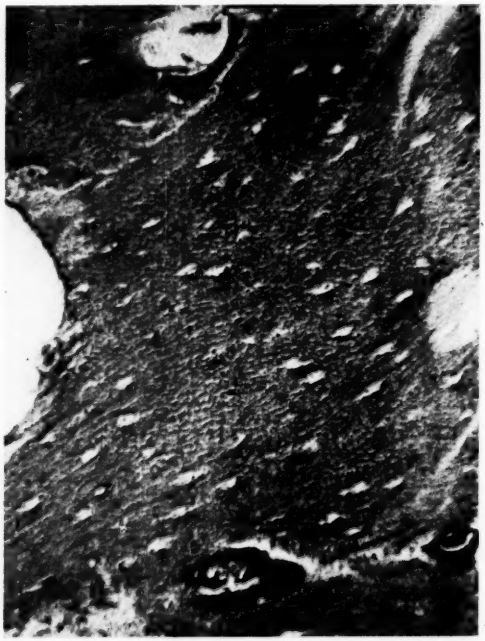

Fig. 2.—Photomicrograph of tissue removed several months after operation from the site of an excised costal cartilage in a patient with pulmonary tuberculosis. This tissue appears to be true bone. The Haversian canals and bone corpuscles are clearly seen.

1. Decrease in Elasticity of the Thoracic Wall.—It has been frequently observed in patients on the chest service of the University of Minnesota Hospitals that, following the partial excision of the costal cartilages together with the costochondral junctions and a short segment of the ribs during the performance of thoracoplasty, the chest wall within a few months regains a rigid condition. X-ray examination of the chests of these patients shows, in addition, that areas of calcification appear at the site of the previously excised cartilages. Furthermore, histologic study of tissues removed from these sites shows the presence of true bone, as indicated in Fig. 2. These findings, combined with the evident rigidity of the chest wall, have given the impression that the excised costal cartilages have regenerated as bone. A method, therefore, would seem to be at hand by which the thoracic walls of the

dog might be made rigid experimentally, if following excision of the costal cartilages, regeneration occurred in the same manner as it appears to occur in man.

Under sodium pentobarbital anesthesia four dogs were operated upon, and all the costal cartilages and costochondral junctions on both sides of the chest from the first rib through the eighth or ninth were removed subperichondrially in four stages. At the time the cartilages were removed, the pseudoarthroses between the costal cartilages and the sternum were curetted until cancellous bone was encountered. The first stage consisted of removing by blunt subperichondrial dissection alternate cartilages on one side of the chest. At the second stage, a week later, the opposite side was operated upon in a similar fashion. Approximately two and one-half months later, the remaining cartilages on each side were removed as before. Four months after the last costal cartilages were removed, the diaphragms were reefed as described below.

2. Increase in Size of the Thoracic Cavity.—An operation on the diaphragm by which it could be shortened in the anteroposterior direction was devised. It was thought that in this manner the diaphragm could be made to assume a position and conformation similar to that seen in emphysema, and that at the same time the volume of the thoracic cavity could be enlarged. This operation was performed as follows: Through an upper abdominal midline incision the tendinous portion of the left half of the diaphragm was grasped with Allis forceps and slowly pulled downward during the inspiratory phase of several respirations. Lembert sutures of silk were then used to make a reef about 1½ inches wide in the muscular anterior portion. This reef was made to extend from the midline of the body to the midline of the axilla. The right side was then treated in a similar manner.

3. General Observations.—To determine the effect on the lungs of these operative procedures on the thoracic walls, measurements of the tidal air and intrapleural pressure were made as in the experiments with tracheal valves. The coefficient of pulmonary distensibility was determined before the beginning of the experiment and again just before sacrifice some weeks later. After sacrificing the animals, their lungs were removed immediately from the chest, inspected grossly, and sections cut for later histologic study.

In some instances, roentgenologic examinations were made both before and after the experiments to determine the effect of the procedures on the level of the diaphragms. In those dogs in which the costal cartilages were resected, the circumference of the thorax was noted at intervals to determine whether or not it increased or decreased.

The lungs of all dogs, whether they died during the course of the experimental period or at the time of sacrifice, were examined grossly and microscopically for the characteristic signs of pulmonary emphysema.

RESULTS

I. Respiratory Obstruction:

a. Obstruction of Expiration.—The results in ten dogs with valves placed in the tracheas so as to obstruct expiration are given in Table I. It will be noted that, in general, the presence of the valve was well tolerated. Two of the animals died of pneumonia after five and seven weeks, respectively. During the experimental period the click of the valve door on the metal stop could be heard easily with a stethoscope. When compelled to exert themselves by running, all the animals became markedly dyspneic. After a few moments they would stop, place their

TABLE I

EFFECT OF INTRATRACHEAL VALVE OBSTRUCTING EXPIRATION MORE THAN INSPIRATION

DOG	BEFORE INSERTION OF VALVE				AFTER REMOVAL OF VALVE				LENGTH OF EXPERIMENT (WK.)	HISTOLOGIC EVIDENCE OF PULMONARY EMPHYSEMA
	TIDAL AIR (C.C.)	INTRAPLEURAL PRESSURE (CM. OF WATER)		COEFFICIENT OF PULMONARY DISTENSIBILITY	TIDAL AIR (C.C.)	INTRAPLEURAL PRESSURE (CM. OF WATER)		COEFFICIENT OF PULMONARY DISTENSIBILITY		
		INSP.	EXP.			INSP.	EXP.			
1	280	−8.00	+0.50	7.50	340	−12.00	+5.00	17.00	18	Gross + Micro. 3+
2	320	−9.50	−1.50	8.00					16	Gross + Micro. 2+
3					320	−14.00	−2.00	12.00	23	Gross − Micro. 1+
4	300	−7.00	−4.00	3.00		−11.50	+3.00	14.50	17	Gross + Micro. 3+
5	300	−11.00	−5.00	6.00	400	−11.00	−1.00	10.00	16	Gross − Micro. 3+
6	320	−11.00	−1.00	10.00	440	−17.00	+1.00	18.00	20	Gross + Micro. 1+
7	340	−11.00	−3.00	8.00	Died of pneumonia				5	Gross − Micro. −
8	160	−10.00	−1.00	9.00	Died of pneumonia				7	Gross − Micro. 1+
9		−12.50	−2.75	9.75	190	−16.25	−3.50	12.75	12	Gross + Micro. 2+
10		−8.50	−0.00	8.50	310	−20.00	−4.50	15.50	8	Gross + Micro. 3+

TABLE II

EFFECT OF INTRATRACHEAL VALVE OBSTRUCTING INSPIRATION MORE THAN EXPIRATION

DOG	BEFORE INSERTION OF VALVE				AFTER REMOVAL OF VALVE				LENGTH OF EXPERIMENT (WK.)	HISTOLOGIC EVIDENCE OF PULMONARY EMPHYSEMA
	TIDAL AIR (C.C.)	INTRAPLEURAL PRESSURE (CM. OF WATER)		COEFFICIENT OF PULMONARY DISTENSIBILITY	TIDAL AIR (C.C.)	INTRAPLEURAL PRESSURE (CM. OF WATER)		COEFFICIENT OF PULMONARY DISTENSIBILITY		
		INSP.	EXP.			INSP.	EXP.			
11	400	−13.00	−5.00	8.00	Died of mediastinitis				10	Gross + Micro. 2+
12	420	−15.00	−7.00	8.00					8	Gross + Micro. 3+
13		−8.00	0.00	8.00	180	−12.00	−3.00	9.00	8	Gross + Micro. 3+
14	190	−9.50	−5.00	4.50					6	Gross + Micro. 3+
15	440	−8.00	−2.00	6.00	Died of pneumonia				6	Gross − Micro. −
16	300	−12.50	−3.00	9.50	160	−11.00	+1.50	12.50	30	Gross + Micro. 3+
17	260	−12.00	−1.50	10.50	Died of mediastinitis				4	Gross − Micro. −
18	260	−8.50	−2.50	6.00		−8.00	−1.50	6.50	15	Gross + Micro. 3+
19	380	−11.00	−1.00	10.00	Died of pneumonia and mediastinitis				4	Gross − Micro. −

forefeet near their hind feet, and with labored effort, attempt to overcome the respiratory resistance. If allowed to remain quiet, however, the dogs showed little evidence of respiratory difficulty.

The coefficient of pulmonary distensibility before the insertion of the valve and after its removal some weeks later shows a definite increase. Such findings are consistent with the development of pulmonary emphysema.

Six lungs showed gross evidence of pulmonary emphysema. They were light and feathery in texture, with occasional small subpleural blebs. Of the remaining four lungs, two were so involved with pneumonia that any signs of emphysema which might have been present were masked.

The microscopic evidence of pulmonary emphysema, consisting of dilatation and rupture of many alveolar walls, with thinning out of those remaining, was definite in six dogs. In three other dogs these findings were present to a minimal extent. Since similar findings are reported to be encountered frequently in the lungs of normal dogs, the significance of these minimal findings is questionable. The lungs of Dog 7, which died of pneumonia, showed no evidence of emphysema on microscopic examination.

X-ray films taken before the insertion of the valves and after their removal, to determine the position of the diaphragm, seemed to indicate in some dogs that the diaphragm was slightly displaced downward. In others no change could be detected.

b. Obstruction of Inspiration.—The findings in those dogs in which valves were inserted so as to obstruct inspiration rather than expiration are presented in Table II. During the experimental period, these animals, when exercised, reacted similarly to the dogs with expiratory obstruction. The valves, however, were less well tolerated. Four of the nine dogs died from either pneumonia or mediastinitis. In only three animals was the coefficient of pulmonary distensibility determined before the insertion of the valve and after its removal. In each case, however, it was increased, apparently as an effect of the valve.

In a few instances optical registrations of the intrapleural pressure and velocity of air flow in the trachea were made to study the effect of the inspiratory obstruction. Fig. 3 is a portion of such registrations made with Dog 13, eight weeks after insertion of the valve.

Roentgenologic examinations to determine any shift in the position of the diaphragm were unequivocal.

The lungs of six dogs showed some evidence of emphysema when examined grossly. The three remaining dogs whose lungs showed no gross changes indicative of emphysema died of pneumonia or mediastinitis a relatively short time after the start of the experiment. On microscopic examination, the lungs of the six animals with emphysematous changes

which could be detected on gross examination also showed definite pathologic pictures consistent with the gross appearance.

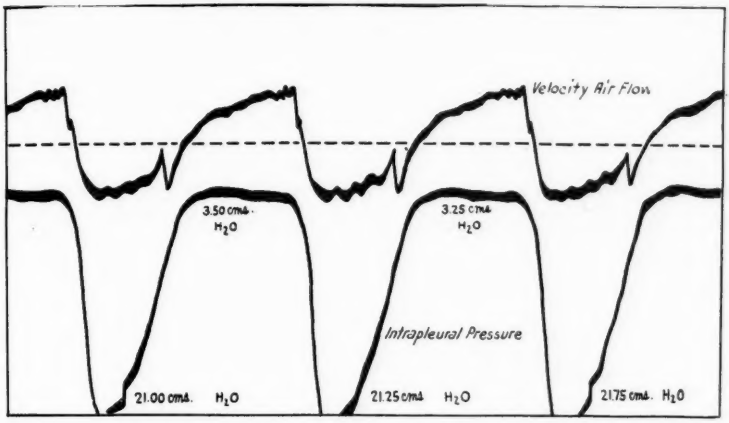

Fig. 3*A*.

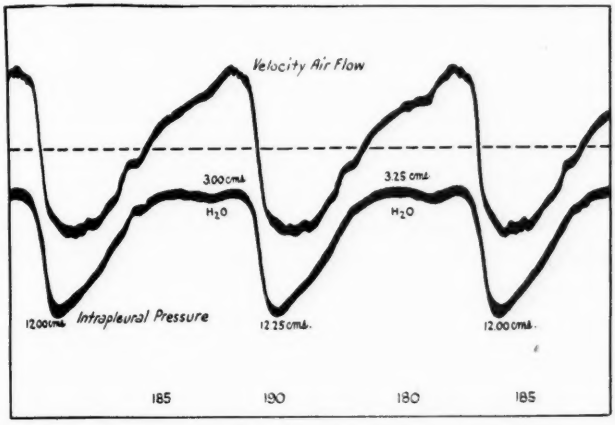

Fig. 3*B*.

Fig. 3.—Photographic tracings of the intrapleural pressure and velocity of air flow in the trachea of Dog 13. These tracings should be read from right to left. *A*, Eight weeks after insertion of a tracheal valve which obstructed inspiration. The effect of the closing of the valve on the velocity of the air flow in the trachea is plainly seen as the notch present in the tracing at the beginning of inspiration. Associated with this is a marked decrease in the intrapleural pressure. *B*, Immediately after removal of the valve and taken about fifteen minutes after *A*. The change in intrapleural pressure during respiration is much less.

c. Control Experiments.—The findings in the three dogs in which valves from which the hinged door had been removed were inserted in the trachea are shown in Table III. It was thought that these animals would serve as controls for the others. To all appearances, the presence of these short metal cylinders within the trachea did not interfere in any way with respiration.

TABLE III

CONTROL SERIES: EFFECT OF INSERTING A CYLINDER IN THE TRACHEA

DOG	BEFORE INSERTION OF CYLINDER				AFTER INSERTION OF CYLINDER				LENGTH OF EXPERIMENT (WK.)	HISTOLOGIC EVIDENCE OF PULMONARY EMPHYSEMA
	TIDAL AIR (C.C.)	INTRAPLEURAL PRESSURE (CM. OF WATER)		COEFFICIENT OF PULMONARY DISTENSIBILITY	TIDAL AIR (C.C.)	INTRAPLEURAL PRESSURE (CM. OF WATER)		COEFFICIENT OF PULMONARY DISTENSIBILITY		
		INSP.	EXP.			INSP.	EXP.			
20	540	−9.50	+1.00	10.50	280	−10.00	+1.50	11.50	9	Gross + Micro. 2+
21	200	−9.00	−0.50	8.50	200	−9.50	+1.50	11.00	9	Gross + Micro. 2+
22	220	−7.50	+0.50	8.00	180	−5.50	−2.00	3.50	9	Gross − Micro. 1+

The coefficient of pulmonary distensibility for these dogs was determined before the insertion of the cylinders and nine weeks later when the cylinders were removed and the animals sacrificed. In two animals there was an increase, and in one, a decrease. Because of the small number of animals, the significance to be attached to these determinations is questionable.

On gross examination the lungs of two dogs showed signs of emphysema to some degree. In the other animal, the lungs appeared to be perfectly normal. These gross findings were substantiated by microscopic examination. In two animals histologic changes were found which were definitely abnormal and which seemed to be indicative of minimal emphysematous changes within the lung parenchyma.

Fig. 4.—Roentgenogram of sternum and regenerated tissue at the site of the costal cartilages excised subperichondrially six months before. There has been marked regeneration of cartilage, but no bone has been formed.

II. Operative Procedures Tending to Enlarge the Thoracic Cavity:

a. Excision of Costal Cartilages.—The results of excision of the costal cartilages were somewhat disappointing. In four dogs in which the cartilages were removed, the thoracic cage, instead of becoming more rigid than before, became definitely less rigid and smaller in circumference. The decrease in the thoracic circumference at the level of the ensiform cartilage five months after the removal of all the cartilages is shown in Table IV.

TABLE IV

CIRCUMFERENCE OF THE THORAX OF DOGS MEASURED AT THE LEVEL OF THE ENSIFORM CARTILAGE BEFORE AND AFTER COSTOCHONDRECTOMY

DOG	BEFORE OPERATION (CM.)	5 MO. AFTER REMOVAL OF COSTAL CARTILAGES (CM.)
23	60	58
24	57	52.25
25	53	50.50
26	55	54

A few weeks following the excision of the cartilages, regeneration began to occur, but the regenerated tissue remained pliable until the death of the animals, approximately six months later. After death, the breast plates of two dogs were removed and examined roentgenologically for evidence of calcification, as indicated in Fig. 4. None was found. Sections of the regenerated tissue were then cut and stained for examination. These sections showed an extensive regeneration of cartilage from the perichondrium which had been left at the time of the chondrectomies.

b. Reefing of Diaphragm.—The effect of reefing on the level of the diaphragm in four dogs is indicated in Table V. This table was constructed from information obtained by determining the position of the domes of the diaphragm with respect to the posterior ends of the ribs on x-ray films of each dog before and immediately after the operation. Since the domes of the diaphragm were displaced caudally about one and one-half intercostal spaces in each case, the volume of the thoracic cavity was enlarged probably to a considerable extent.

TABLE V

CHANGE IN POSITION OF THE DIAPHRAGM AFTER ANTERIOR REEFING

DOG	SIDE	BEFORE OPERATION	AFTER OPERATION
23	Right	At 10th rib	Between 11th and 12th ribs
	Left	Between 9th and 10th ribs	At 12th rib
24	Right	Between 8th and 9th ribs	Between 10th and 11th ribs
	Left	Between 9th and 10th ribs	Between 11th and 12th ribs
25*	Right	Between 9th and 10th ribs	Between 11th and 12th ribs
	Left	Between 10th and 11th ribs	Between 11th and 12th ribs
26*	Right	A 9th rib	Between 10th and 11th ribs
	Left	Between 9th and 10th ribs	Between 10th and 11th ribs

*These dogs had both phrenic nerves cut one week before the diaphragm was reefed.

In Table VI are shown the results of reefing the diaphragms of seven dogs on the intrapleural pressure and the coefficient of pulmonary distensibility. Dogs 24 and 26, which died shortly after the operation on the diaphragm from respiratory embarrassment, are not included in the table. Dogs 23 and 25 had had their costal cartilages excised previously, as described above.

TABLE VI

EFFECT OF REEFING DIAPHRAGM

DOG	BEFORE REEFING DIAPHRAGM				AFTER REEFING DIAPHRAGM				LENGTH OF EXPERIMENT (WK.)	HISTOLOGIC EVIDENCE OF PULMONARY EMPHYSEMA
	TIDAL AIR (C.C.)	INTRAPLEURAL PRESSURE (CM. OF WATER)		COEFFICIENT OF PULMONARY DISTENSIBILITY	TIDAL AIR (C.C.)	INTRAPLEURAL PRESSURE (CM. OF WATER)		COEFFICIENT OF PULMONARY DISTENSIBILITY		
		INSP.	EXP.			INSP.	EXP.			
23	340	−8.50	0.00	8.50	140	−11.00	−2.00	9.00	19	Gross + Micro. 2+
25	240	−13.00	−3.00	10.00	240	−18.00	−6.00	12.00	18	Gross − Micro. 2+
27	260	−9.50	−4.50	5.00	360	−10.50	+1.00	11.50	23	Gross + Micro. 3+
28	260				360	−9.00	+1.00	10.00	25	Gross + Micro. 3+
29		−11.50	−5.25	6.25		−14.00	−4.50	9.50	24	Gross + Micro. 3+
30		−5.00	−0.50	4.50		−18.00	−1.50	16.50	24	Gross + Micro. 3+
31		−8.00	−2.00	6.00		−10.25	−2.50	7.75	16	Gross + Micro. 2+

It should be noted that the coefficient of pulmonary distensibility was consistently elevated following the reefing operation, indicating that in some way the elastic properties of the lungs had been altered.

On gross examination of the lungs, only one of the six animals did not present evidence of emphysematous changes. When examined microscopically, all the lungs were found to contain unmistakable fragmentation and atrophy of the alveolar walls, with the formation of large dilated air spaces. These histologic changes, in general, were more well defined and of greater degree in this group of dogs than in any other. Frequently the emphysematous changes were found in sections taken from the center of the lobes, but usually the most marked changes were in the immediate subpleural areas.

DISCUSSION

Before an accurate appraisal of the histologic evidence of the presence of emphysema in the lungs of dogs can be made, one must be familiar with the variations in the histologic picture of normal lungs. To evaluate the histologic changes found in the lungs of dogs reported in this paper, four to six sections taken immediately after death from various parts of the lungs of eight dogs that died as a result of lesions not affecting the walls of the thoracic cavity or its contents have been studied. The lungs of these animals frequently showed areas in which the alveoli appeared to be enlarged two to three times the average size. In addition, occasionally the alveolar walls appeared to be broken. Since these findings may be present in the normal lung, the investigator must be careful in attributing similar changes in other lungs to any experimental procedure. In the experiments reported in this paper, the lungs in which it is indicated that emphysematous changes of grades 2+ and 3+ were present exhibited a histologic appearance which could not be accounted for on the basis of the normal variation. Certain lungs have been reported as 1+ for the presence of emphysema. In these animals the histologic picture, while suggestive of pathologic change, may have shown only an unusual degree of normal variation.

Were the histologic changes the sole basis for deciding whether or not emphysematous changes had been produced in the lungs of our experimental animals, the rather definite conclusions reached might possibly be subject to criticism despite the critical attitude which has been taken. In general, however, these findings were consistently corroborated by the coefficients of pulmonary distensibility.

The results of the experiments here reported indicate that changes in the lungs of dogs identical with those found in patients with pulmonary emphysema can be produced by obstructing the tidal flow of air through the trachea, as well as by reefing the anterior portion of the diaphragm.

The production of a minimal degree of emphysema in two of three dogs in which cylinders which only slightly encroached upon the lumen

were placed in the trachea casts some doubt on the importance of expiratory or inspiratory obstruction when obtained by intratracheal valves in experimental animals.

It would seem rational to suppose that the ultimate cause of rupture of the alveolar walls in emphysema must be some stretching force, and this can only be the stress placed on them during the inspiratory phase of respiration. Since, normally, the alveolar walls tend to remain intact throughout life, the conditions which are necessary for the development of emphysema must either increase the stresses of inspiration or alter the strength of the alveolar walls so that normal stresses can no longer be withstood. In emphysema it is quite possible that both these things occur.

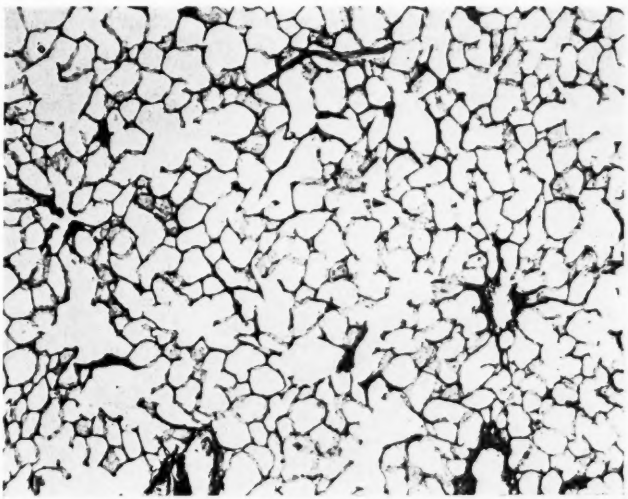

Fig. 5.—High-power photomicrograph of a portion of the lung of a normal dog.

The thoracic girth of dogs has been found by Schall to be increased in both inspiratory and expiratory obstruction. As a corollary it follows that the size of the lung and the tension of the alveolar walls must be increased. Cloetta and Chillingsworth and Hopkins have determined that distention of the lungs tends to decrease the caliber of the pulmonary capillaries and decreases the flow of blood through them. This decrease in blood flow is further reinforced in the presence of expiratory obstruction by an increase in the intra-alveolar air pressure which will tend to collapse the capillaries already narrowed by distention. Under such conditions nutritive disturbances and atrophy of the alveolar walls could easily occur. The stress of inspiration on the alveolar walls might then produce emphysema. In the presence of inspiratory obstruction, slightly different conditions prevail. The lungs become enlarged through

violent inspiratory efforts. The blood flow through the pulmonary capillaries is decreased by the overdistention of the lung, as in expiratory obstruction. This decrease in blood flow, however, is probably less than that which occurs with expiratory obstruction, for the intra-alveolar air pressure is decreased instead of increased. On the other hand, inspiration is much more forceful and the stresses thereby produced in the alveolar walls are greater. The total effect of these factors may well be the same whether inspiration, expiration, or both are impeded.

The results obtained in those dogs in which the diaphragm was reefed indicate that the elastic properties of the lungs can be decreased with the production of emphysematous changes in the lung parenchyma in the absence of any respiratory obstruction. The question naturally arises as to what is the causative factor in the production of these changes. To answer this question definitely, further studies will be necessary.

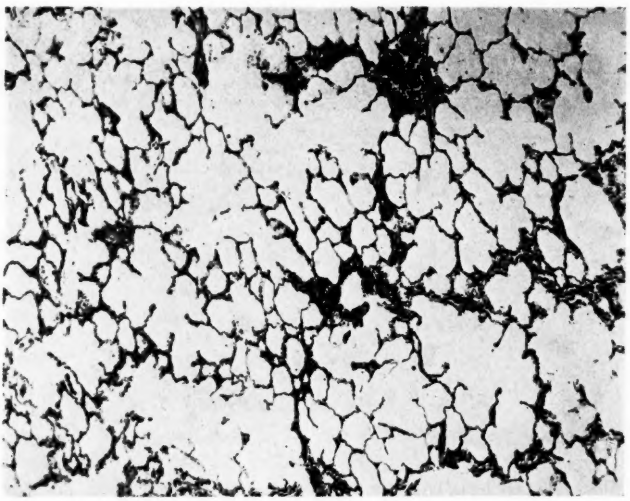

Fig. 6.—High-power photomicrograph of a portion of the lung of a dog which had a valve in the trachea obstructing expiration for twelve weeks. A patchy type of emphysema was present, with atrophy and rupture of the alveolar walls.

Although the costal cartilages were excised in four dogs, only two are included in Table VI because the other two died a few days after the diaphragm was subsequently reefed. It seems unreasonable to suppose that costochondrectomy can have had any positive influence in the emphysema produced in the two surviving dogs. The result of this procedure was to increase the flexibility of the thoracic wall and decrease the volume of the thoracic cavity, conditions which are directly opposed to those found in all cases of emphysema occurring in the human lung.

The consistent production of emphysema following reefing of the diaphragm, with its assumed accompanying increase in the size of the thoracic cavity, is an important finding and tends to confirm Loescheke's theory.

It may rationally be assumed that the emphysema produced by enlarging the thoracic cavity occurs in the same manner as it was presumed to occur with respiratory obstruction. The overdistention of the lungs tends to decrease the blood flow through the pulmonary capillaries, which leads to atrophic changes in the alveolar walls. These, in turn, are acted upon by unusual stresses during inspiration because of the increased distention of the lungs.

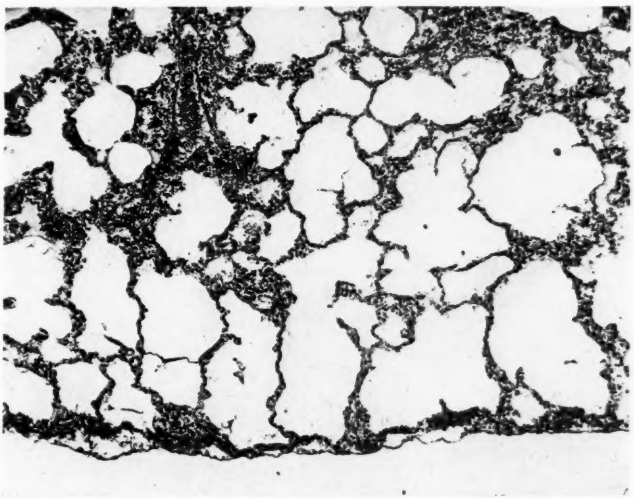

Fig. 7.—High-power photomicrograph of a portion of the lung of a dog which had a valve in trachea obstructing inspiration for eight weeks. In this dog the marginal emphysema was quite striking.

It is difficult, however, to reconcile the production of emphysema by an increase in the size of the thorax with the opinions of Rienhoff and Bremer. These men, among others, now maintain, and consider the question settled, that the compensatory emphysema which follows lobectomy or pneumonectomy is not a true emphysema with rupture of the alveolar walls, but a general dilatation of all the alveolar air spaces. Rienhoff, Reichert, and Heuer, in 1935, reported their results of a careful histologic study of the pulmonary tissue remaining in ten dogs after pneumonectomy. These investigators found no signs of true emphysematous changes several months after pneumonectomy. On the other hand, lobectomy when performed experimentally in dogs by Adams and Livingstone in 1932, and by Kountz, Alexander, and Prinzmetal in 1936, was found to be regularly followed by true emphysema.

One point should be made clear. The experiments mentioned above have all involved the removal of portions of the pulmonary parenchyma,

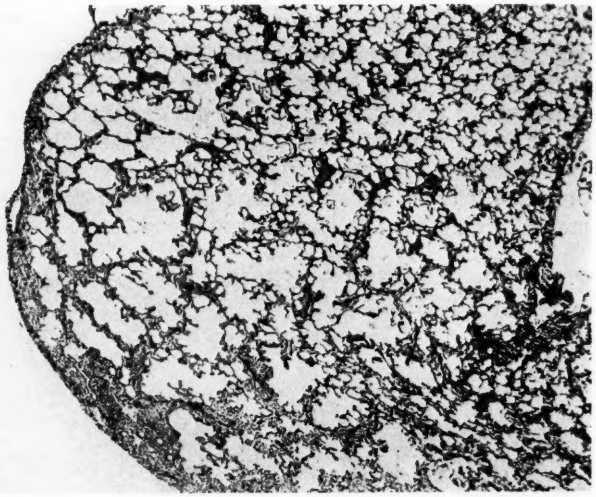

Fig. 8*A*.—Low-power photomicrograph of a portion of the lung of a dog six months after costochondrectomy and reefing of the diaphragm. A marked degree of emphysema is present.

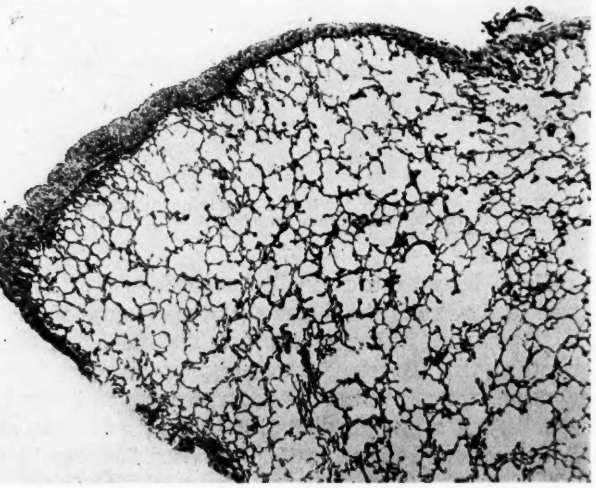

Fig. 8*B*.—Low-power photomicrograph of a portion of the lung of a dog one month after reefing of the diaphragm. There is an extensive rupture of the alveolar walls throughout the lung.

so that after the removal, the proportional amount of blood flow through each lobe of lung remaining has been greater than before. In the ex-

periments reported in this paper, in which the diaphragm has been reefed, the proportional amount of blood flow through each lobe of lung has remained the same. Whether or not this circumstance makes any difference in the development of emphysema must be demonstrated by further studies. The fact remains that dogs in which the diaphragm was reefed in the manner previously described showed definite changes in the parenchyma of the lung which closely simulated those found in the human being afflicted with true pulmonary emphysema.

It has long been held by many clinicians that pulmonary emphysema eventually produces a hypertrophy of the right ventricle, and that death in patients with emphysema is frequently due to dilatation and failure of the right side of the heart. These ideas were brought to mind when it was noted at the post-mortem examination of a dog that died following reefing of the diaphragm that the right ventricle was unusually dilated.

The hearts of twenty-five normal dogs and six other dogs in which emphysema was produced by reefing the diaphragm were studied by the method of Dr. G. F. Higgins, to determine if any hypertrophy of the right ventricle had occurred. The hearts were first hardened in a 1 per cent solution of formaldehyde. The auricles then were trimmed away, and the coronary blood vessels, together with the epicardial fat, were removed from the surface of the ventricles. With a sharp knife, transverse sections of the ventricles 1 cm. in thickness were cut. In such sections the decussation of the muscle fibers forming the ventricular walls can be seen easily. With this as a guide, the right ventricle was cut away from the left ventricle and the weight of each was obtained.

The ratio of the left ventricle to the right in the dogs with hearts considered to be normal was found to vary from 1.30 to 1.80, with an average of 1.58. In the six dogs in which pulmonary emphysema was produced by reefing the diaphragm, this ratio varied from 1.42 to 1.77, with an average value of 1.62. It can be said, therefore, that no hypertrophy of the right ventricle occurred in the experimental animals thus studied.

SUMMARY

Despite years of research and volumes of speculation, the essential nature of pulmonary emphysema remains unsolved. The theories of etiology which merit consideration can be classified under two general heads: (1) Some type of obstruction to respiration; and (2) some change in the form or function of the walls of the thoracic cavity. If it can be shown that emphysema is the same end result of different pathologic processes, much will be accomplished toward reconciling the varied theories of its production, clarifying its essential nature, and establishing a rational basis for its treatment.

In the past, several investigators have claimed to have produced true pulmonary emphysema in laboratory animals by various methods. The

methods employed have been concerned with one or more of the following principles:

1. Obstruction of some type to the passage of air through the tracheobronchial tree.
2. Increase in the space which functioning lung tissue is made to occupy.
3. Decrease in the oxygen tension of the air breathed.

By each method the midcapacity of the thorax has been increased, and consequently, the average stress borne by the alveolar septa of the lungs has been increased.

The investigations here reported are concerned with the study of the effect on the elasticity of the lung and the histologic changes in the pulmonary parenchyma incident to respiratory obstruction and to operative procedures which tend to enlarge the thoracic cavity. Respiratory obstruction was obtained by the insertion of special valves into the tracheas of dogs. In ten dogs expiration was obstructed, and in nine dogs, inspiration. After intervals varying from four to thirty weeks, these animals were sacrificed.

On the basis of histologic study, as well as changes in the coefficient of pulmonary distensibility, pulmonary emphysema was found to have been produced in six of the ten dogs in which expiration was obstructed and in six of the nine dogs in which inspiration was obstructed. In two of three control dogs in which valves without flaps were inserted for nine weeks, minimal emphysematous changes were found.

In an attempt to produce a rigid thorax, the costal cartilages were resected in four dogs. These cartilages regenerated and the thorax was less rigid than before. In these animals, as well as in five other dogs, the anterior portion of both halves of the diaphragm was reefed. At sacrifice sixteen to twenty-five weeks later, all seven dogs which survived the reefing operation were found to have evidence of pulmonary emphysema.

CONCLUSIONS

Emphysematous changes in the pulmonary parenchyma occur in dogs within four to thirty weeks after valves which obstruct primarily either inspiration or expiration are placed in the trachea.

Similar changes are produced within eighteen to twenty-five weeks if the anterior portion of the diaphragm is reefed.

Costal cartilages of dogs, when resected subperichondrially, regenerate within six months as true cartilage.

It is quite probable that any process which increases the average size of the thorax for a sufficient length of time will produce the pathologic changes in the lung parenchyma and the clinical entity known as pulmonary emphysema.

REFERENCES

1. Adams, W. E., and Livingstone, H. M.: Lobectomy and Pneumectomy in Dogs, Arch. Surg. **25**: 898, 1932.
2. Bremer, J. L.: The Fate of the Remaining Lung Tissue After Lobectomy or Pneumonectomy, J. THORACIC SURG. **6**: 336, 1936-37.
3. Brown-Sequard: Indication d'un Mode Nouveau de Production de l'Emphysème Pulmonaire, Compt. Rend. Soc. de biol. **37**: 354, 1885.
4. Bullara, L.: Enfisema Pulmonare da Occlusione Nasale e. Ana Patogenesi, Riforma med. **3**: 387, 1900.
5. Campbell, J. A.: Note on Some Pathological Changes in the Tissues During Attempted Acclimatization to Alterations of O_2 Pressure in the Air, Brit. J. Exper. Path. **8**: 347, 1927.
6. Chillingsworth, F., and Hopkins, R.: Physiologic Changes Produced by Variations in Lung Distension, Am. J. Physiol. **51**: 289, 1920.
7. Christie, R. V., and McIntosh, C. A.: The Measurement of the Intrapleural Pressure in Man and Its Significance, J. Clin. Investigation **8**: 279, 1934.
8. Cloetta, M.: Über die Zirkulation in der Lunge und deren Beeinflussung durch Über-und Unterdruck, Arch. f. exper. Path. u. Pharmakol. **66**: 409, 1911.
9. Cloetta, M.: Untersuchungen über die Elastizetat der Lunge und deren Bedeutung für die Zirkulation, Arch. f. d. ges. Physiol. **152**: 339, 1913.
10. Freund, W. A.: Krankungen des Knorpeligen Thorax auf Entstehung gewisser Lungenkrankheiten, Verhandl. d. phys. med. Gesellsch. **9**: 223, 1859.
11. Freund, W. A.: Zur operativen Behandlung gewisser Lungen Krankheiten insbesondere des auf starrer Dilatation beruhenden alveolären Lungenemphysems (mit Operationsfall), Ztschr. f. exper. Path. u. Therap. **3**: 479, 1906.
12. Friedman, E. D., and Jackson, H. C.: The Carbon Dioxid Content of Blood and of Alveolar Air in Obstructed Expiration, Arch. Int. Med. **19**: 767, 1917.
13. Harris, W. H., and Chillingsworth, F.: The Experimental Production in Dogs of Emphysema with Associated Asthmatic Syndrome by Means of an Intratracheal Ball Valve, J. Exper. Med. **30**: 75, 1919.
14. Hinshaw, H. C.: Experimental Production of Chronic Pulmonary Emphysema in Animals, Proc. Staff Meet., Mayo Clin. **13**: 599, 1938.
15. Hirtz: Quoted by Schall, Thèse de Paris, 1878.
16. Köhler, H.: Ueber die Compensation Mechanischer Respiration sstönungen und die physiologische Bedeutung der Dyspnoe, Arch. f. exper. Path. u. Pharm. **7**: 1, 1877.
17. Kountz, W. B., and Alexander, H. L.: Non Obstructive Emphysema, J. A. M. A. **100**: 551, 1933.
18. Kountz, W. B., Alexander, H. L., and Prinzmetal, M.: The Heart in Emphysema, Am. Heart J. **11**: 163, 1936.
19. Laënnec, R. T. H.: A Treatise on the Diseases of the Chest and on Mediate Auscultation. Translation by J. Forbes, New York, Samuel S. Wood and W. Wood, 1838, p. 161-185. (Medicine **13**: 251, 1934.)
20. Loeb, L. M.: The Etiology of Emphysema, Arch. Int. Med. **45**: 646, 1930.
21. Loeschcke, H.: Ueber Wechselbeziehungen Zwischen Lunge und Thorax bei Emphysem, Deutsche med. Wchnschr. **37**: 916, 1911.
22. Loeschcke, H.: Handbuch der Speziellen Pathologischen Anatomie und Histologie, Berlin, 1928, Julius Springer, vol. 3, p. 612.
23. Nissen, R.: Experimentelle Untersuchungen zur Theorie der Entstehung des Lungen-emphysems, Deutsch. Ztschr. f. Chir. **200**: 177, 1927.
24. Prinzmetal, M.: The Relation of Inspiratory Distention of the Lungs to Emphysema, J. Allergy **5**: 493, 1933-34.
25. Rienhoff, W. F.: Intrathoracic Anatomical Readjustments Following Complete Ablation of One Lung, J. THORACIC SURG. **6**: 254, 1936-37.
26. Rokitansky: See Laënnec.
27. Schall, H.: Experimentelle Beiträge zur Entstehung des Lungen-emphysems, Beitr. z. Klin. d. Tuberk. **14**: 407, 1909.
28. Sudsuki, K.: Ueber Lungen-emphysems, Arch. f. path. Anat. u. Physiol. u. f. klin. Med. **157**: 438, 1899.

NOTE ON THE POSITION OF THE PATIENT IN THE PERFORMANCE OF THORACOPLASTY AND CERTAIN OTHER OPERATIONS

HUGH C. TRUMBLE, F.R.C.S.
MELBOURNE, AUSTRALIA

SINCE the year 1885, in which Cerenville of Lausanne performed an operation that may be regarded as the first "thoracoplasty," surgeons have gradually developed the modern operation of posterolateral extrapleural thoracoplasty. At the present time there is more or less general agreement upon the broad principles of the operation, but there is still room for improvement in detail. In this brief communication it is proposed to describe certain technical procedures which may be of interest.

Usually, the various stages of the operation are performed with the patient lying on his side, with his diseased lung uppermost. This position appears to be a bad one, for several reasons. Pus from the diseased lung tends to run into the large bronchi and may easily gravitate or be aspirated into the sound lung. The mediastinum sags downward and encroaches upon the space occupied by the healthy lung, and this effect becomes more pronounced later in the operation when extensive sections of ribs have been removed. Ventilation of the lung may be impeded, and the action of the heart embarrassed. Lastly, during the performance of the upper stages of the operation, the scapula must be elevated actively in order to provide adequate access.

It will thus be seen that the force of gravity acts potently to the detriment of the patient and the embarrassment of the surgeon.

In order to eliminate the disadvantages enumerated above, for the past nine years I have operated with the patient lying in the lateral prone position, with the healthy lung uppermost (Figs. 1 and 2). The patient's lower arm and shoulder pass below the level of the table, through a gap which may be provided in one of two ways. Many tables have a divided foot section; one half is dropped. The thorax rests firmly on the middle section of the table, and the head on the undropped half of the foot section. The arm and shoulder project through the gap left by the dropping of the other half of the foot section.

Should the foot section of the table not be so accommodating, a headrest may be used to support the head, the arm projecting through the gap between the headrest and the table.

The position is one of stability and comfort. Respiration is not impeded. Pus which may be present in the diseased lung can scarcely run uphill into the main bronchi, and there is no fear that it may enter

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the sound lung. The mediastinum tends to sag toward the unhealthy lung, so that the space occupied by the healthy lung is increased rather

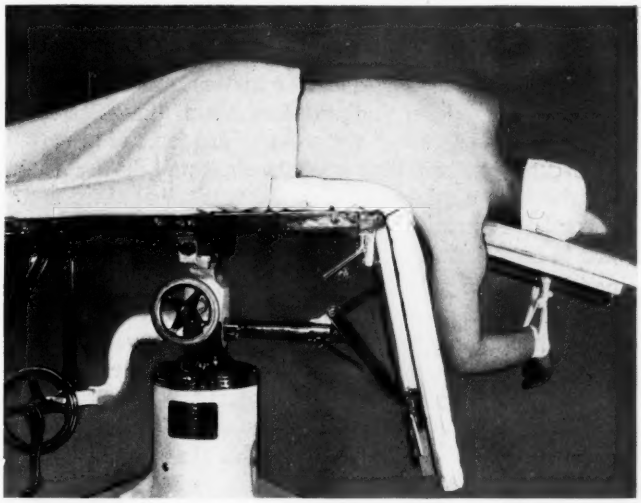

Fig. 1.—Photograph showing the position in which a patient is placed for thoracoplasty. The table has a divided foot section, one half of which has been dropped. The head rests on the other half. Note that the underarm hangs below the level of the table and that its weight tends to drag the scapula away from the spine.

Fig. 2.—Photograph showing the position of the patient as seen from the head end of the table. Note the semiprone position and the way in which the arms are supported.

than decreased. After division of the muscles passing from the spine to the scapula, the latter is dragged away from the spine by the weight

of the arm and shoulder. The effort required to retract the scapula from the chest wall so as to expose the upper ribs is thereby reduced to a minimum. Access is such that it is possible to remove adequate lengths of the uppermost ribs through a much shorter incision than is usually employed. In order to do this, special instruments, whose main virtue is their unusual length (about 12 inches), are used. When sections of the ribs have been removed, flapping of the chest wall is reduced to a minimum because of the action of the force of gravity, which helps to prevent excessive falling in of the chest wall during inspiration. Shock is reduced to a minimum, with the result that there is no need for haste, and the operation may be performed deliberately and with due regard to hemostasis.

The position of the patient described above may be used with advantage in the performance of certain other operations. It is particularly useful when an empyema is to be drained, the patient, of course, being placed with his healthy side uppermost. For operations upon the kidney and ureter, the position is most suitable. A small firm cushion should be inserted under the patient at the waist level. Placed so, the patient is much more comfortable and stable than is usually the case. All danger of compression of the underarm or of the axillary vessels and nerves is eliminated.

THE ROLE OF BRONCHOSCOPY IN THE TREATMENT OF PULMONARY ABSCESS

CHEVALIER L. JACKSON, M.D., AND A. R. JUDD, M.D. (BY INVITATION),
PHILADELPHIA, PA.

THE present-day picture in the treatment of nontuberculous pulmonary abscess is yet in a somewhat indefinite state as evidenced by the recognized high mortality and morbidity rates resulting therefrom. It is acknowledged that a certain number of such abscesses resolve spontaneously, that a certain additional number yield to appropriate nonsurgical treatment, including bronchoscopic drainage, and that still others must be treated surgically. No clear point of demarcation exists in the course of the disease designating where one procedure should be abandoned and another be adopted, either singly or in combination. It is our purpose in the present survey to study particularly bronchoscopic measures as one phase of the treatment of abscess of the lung; and to attempt to establish their relationship to the problem as a whole.

We believe that there exists a definite place for bronchoscopy in the treatment of pulmonary abscess, aside from its value in the diagnosis. However, the time element is important, and bronchoscopy should not be carried past the optimum time for surgical drainage. Goldman,[8] in 1936, pointed out the value of bronchoscopy in diagnosis, in localization of the diseased segment, and also in finding and removing causes when possible. He also values it as a means of determining the state of a patient's pulmonary condition following surgical operation. There are many others[1-10] who recognize bronchoscopy as an important factor in either diagnosis or treatment of lung abscess, or in both. Nevertheless, there seems to be little crystallized opinion regarding the exact role of bronchoscopy in relation to other procedures used. Where there exists a wide diversity of opinion as regards any method, we feel that thorough critical analyses are of value, without necessarily rendering the problem controversial.

In the selection of our cases we have attempted to choose those of patients who have presented themselves at the clinic with a single abscess cavity as evidenced by thorough roentgenologic examination, and those cases which, although showing multiple or multilocular cavities, present satisfactory evidence that the original lesion was *singly* cavernous. Of the cases thus selected, each has been analyzed as to etiology, duration of symptoms prior to entering the clinic, bacteriologic studies, therapeutic procedures, and results. We realize that for a lesion to show a definite cavity upon x-ray examination, there must have been

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drainage of some kind established at some time or another. Such drainage is usually established through a connecting bronchus. We realize also that this eliminates from consideration a number of instances of suppurative pneumonitis, some of which resolved with or without bronchoscopic aspiration and without true abscess formation. It also eliminates some true abscesses that may have ruptured directly into the pleural cavity, thus resulting in a putrid empyema with or without an accompanying pneumothorax. These latter usually occur in the very acute stage and usually before even a diagnostic bronchoscopy has been performed.

The use of iodized oil in the diagnosis of pulmonary abscess is not always accurate. If, when it is instilled into the bronchus of the affected side, it enters the cavity, the result, of course, is highly satisfactory. Still, if it fails to enter a cavity otherwise visualized on the roentgenologic film, one cannot deny the diagnosis. The inflammation and edema around the mouth of the draining bronchus usually do not permit the entrance of the oil, and filling of the cavity proper is impossible. Therefore, we have to base a positive diagnosis of abscess upon the patient's history, the roentgenologic findings, and failure to obtain acid-fast organisms upon appropriate laboratory study of sputum, secretions, or both. The difficulty in the filling of a true abscess cavity with lipiodol has been encountered previously by others, including Furstenberg and Hickey[6] and Mosher.[20] The technique of bronchography employed by us at present corresponds to the catheter method as already described by Jackson and Bonnier.[11]

Definition.—It has been observed for some time past that the term "pulmonary abscess," "abscess of the lung," "lung abscess," and other synonyms have come to be applied rather indiscriminately. They have been used either to designate or describe certain conditions that have not and may never develop into a true abscess. We have already stated that in this survey we have included only those cases in which by x-ray or bronchography there has been demonstrated a single cavity, at the outset at least, known to be nontuberculous. We also state that by so doing we realize that we may have eliminated from consideration certain cases which could have had all the pathologic characteristics of abscess, but yet which could not be demonstrated conclusively as such. This policy has been followed because it is felt that there exist definite differences in the underlying principles of therapy of true abscess of the lung and other conditions to which that term has been inaccurately applied on so many occasions.

It is the opinion of the authors that the term "pulmonary abscess" should be reserved solely for that condition of nontuberculous, cavernous, pulmonary, suppurative disease in which a single open cavity has been adequately demonstrated at the outset, even though at a later date

cavities may become multiple, multilocular, or both. This, then, stands out in contradistinction to the following:

Bronchiectatic abscess, which implies there are one or more abscesses demonstrated to be present, but definitely in conjunction with an underlying primary bronchiectatic disease, based pathologically on the progressive dilatation of the terminal bronchi.

Suppurative pneumonitis, an inflammatory suppurative disease of nontuberculous origin involving the lung parenchyma. This process may progress, producing local tissue destruction, thus becoming a true abscess. However, not until that stage is reached do we have a true abscess. Before this stage, the process is capable of resolution and healing without true abscess formation.

Pulmonary atelectasis, "imperfect expansion," not a true disease entity in itself, but generally a sign of some underlying abnormal condition such as bronchial obstruction by foreign body, neoplasm, etc. The subsequent sequence of events may lead up to abscess formation, bronchiectasis, suppurative pneumonitis, etc., but care should be exercised to distinguish this from actual abscess. Plugs of tenacious sputum constitute the most common cause of obstructive atelectasis. The relief of such obstruction permits free ingress and egress of air, with a return of the lung to its normal degree of aeration.

The confusing of these conditions and the conscious or intentional grouping of them as of one class, "pulmonary abscess" or "pulmonary suppuration," we feel, should be a matter for careful attention, with a view toward eliminating the confusion or at least the looseness that has been occurring in the use of the term "abscess."

Etiology.—The etiology of pulmonary abscess in our series of cases conforms to general opinions already established. It will be noted in Table I that forty-nine cases have followed some form of surgical operation. Of this group, the largest number are post-tonsillectomic. It is also significant to note in this table that only three cases are positively known to have occurred following local anesthesia. The remaining postoperative cases are variously distributed. Next in frequency is that group of forty-six cases in which symptoms date back to some incident of the respiratory system which has been variously diagnosed as "pneumonia," "grippe," "cold," "flu," etc. It is undoubtedly true that many of these cases have had their onset in some vague and insidious manner, and that the symptoms leading to these varied diagnoses are, in fact, the early symptoms at or immediately preceding actual abscess formation. It seems reasonable to suggest that such abscesses may result from a preceding atelectasis of a certain lung segment, brought about by an obstructing mechanism such as tenacious secretion, foreign body, or an inflammatory or stenotic change in the segmental bronchus. One must remember that the lumen of these segmental bronchi is not very large, and therefore it must be realized that an obstruction does not necessi-

TABLE I

ETIOLOGY (124 CASES)

POST-TONSILLECTOMIC		OTHER POSTOPERATIVE		RESPIRATORY		POST-TRAUMATIC		MISCELLANEOUS CAUSES	
General anesthesia	20	General surgery		"Pneumonia"	15	Bullet wound	1	Foreign body	6
Local anesthesia	3	General anesthesia	11	"Colds" and "grippe"	27	Auto accident	1	Carcinoma	1
Unknown anesthesia	12	Postdental	2	"Asthma"	1	Fractured jaw	1	Infected congenital cyst	2
T. & A. contributing cause				Acute sinusitis	1			Aspirated vomitus	1
(Ton. snare as fbdy.)	1			Acute tonsillitis	2			Unknown or indefinite	16
Total	36	Total	13	Total	46	Total	3	Total	26

TABLE II

BACTERIOLOGIC STUDIES OF BRONCHOSCOPICALLY ASPIRATED SPECIMENS OF SECRETIONS

PNEUMOCOCCUS	N. CATARRHALIS	STREPTOCOCCUS		STAPHYLOCOCCUS		SPIROCHETE COMPLEX		MISCELLANEOUS ORGANISMS	
50	50	Hemolyticus	34	Aureus	16	Spirochetes	21	*N. sicca*	2
		Nonhemolyticus	51	Albus	31	Vibrio type	4	*H. influenzae*	8
		Viridans	18	Hemolytic	14	Fusiform bacilli	8	Friedländer's bacillus	3
								Diphtheroids	12
								Bacteroides	9
								B. proteus	1
								M. tetragenus	1

Secretions were examined in 113 cases (91.1 per cent). Only specifically identified organisms are herein included. Sputum examinations are not included in the above table, as such examinations do not represent uncontaminated bronchial secretions, because of the presence of mouth organisms.

tate the presence of a large mass of material. The progressive toxemia arising from the suppurative process distal to this obstruction accounts for the symptoms of which the patient first complains. It is certainly true that in some of the cases the abscess may actually be a sequel to any one of the above-mentioned acute respiratory infections. The remaining (twenty-nine) cases are widely distributed etiologically, their apparent causes including known foreign body, infected lung cyst, mediastinal abscess of foreign body origin (fishbone), trauma, and carcinoma.

BACTERIOLOGY

On all but the earlier cases we have completed bacteriologic studies, using either the sputum or the bronchoscopically aspirated secretions or both. As the sputum studies are not exclusively representative of the flora in the infected lower respiratory system, reference will not be made to them. Bronchoscopically aspirated secretions, on the other hand, do contain a representative flora, and, therefore, an adequate study of them may be considered justified.

Of our 124 patients finally selected, 113 (91.1 per cent) have had thorough study made of their aspirated secretions. The remaining eleven (8.9 per cent) patients include those in whom only sputum examinations were made and those earlier cases on which complete studies are not available. The results have been tabulated and are recorded in Table II.

Summarizing the results of the study, it is felt that the following represents justifiable conclusions in the various pulmonary suppurative entities encountered:

1. In lung abscess, anaerobic and aerobic cultures characteristically show hemolytic staphylococcus, nonhemolytic streptococcus, hemolytic streptococcus, pneumococcus, or *N. catarrhalis.*

2. In pulmonary gangrene, the aerobic culture shows no growth and nothing of special significance. The anaerobic culture shows an admixture of proteolytic anaerobic organisms including spirochetes. These are definitely secondary invaders, but will assume primary importance in an area poorly drained, that is, a "closed area of suppuration." For these there is no known available and specific therapy, except in the case of the spirochetes.

3. Empyema seems to be caused, in its early stages, by the same group of organism found in abscess. When it becomes putrid, however, the flora changes over to that characteristic of gangrene or bronchiectasis. Incidentally, the flora of bronchiectasis is not constant and characteristic. Either no growth or possibly streptococci may be found on aerobic culture. Nonhemolytic streptococci, bacteroides, and fusiform bacilli are typical anaerobic findings.

The only specific therapy available for employment against these various organisms is arsphenamine, or neoarsphenamine in the case of the spirochetes. Sulfanilamide generally is not effective against the

anaerobic hemolytic streptococcus. The only avenue of attack, therefore, lies in the principle of adequate and immediate drainage, either bronchoscopically, combined with postural drainage, or by accepted surgical procedures.

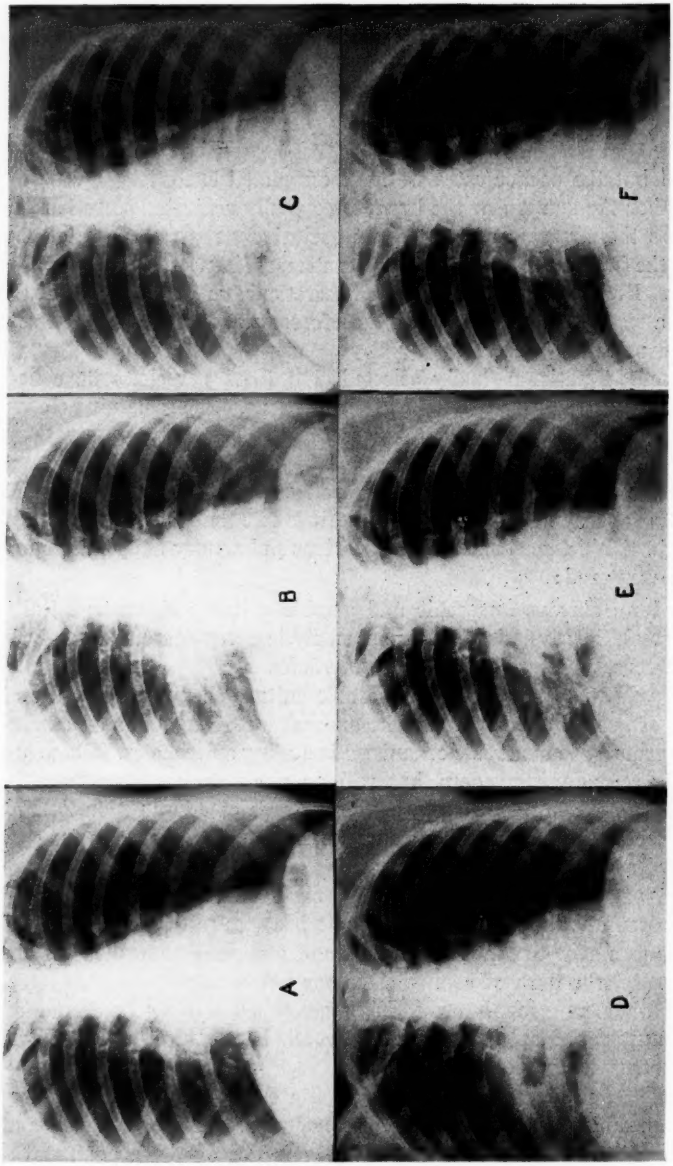

Fig. 1.—Case of post-tonsillectomic abscess in a young woman showing complete spontaneous healing within six weeks. The first film (*A*) was taken three days, the second film (*B*) fourteen days, and the third film (*C*) nineteen days after the tonsillectomy. The abscess reached its maximum excavation in sixteen days (*D*), and then after spontaneous evacuation, rapidly healed as shown by *E* and *F*.

Duration of Symptoms.—Most of the patients with pulmonary abscess encountered in the Temple University clinic have gone through the acute stage and are seen after the abscess has become subacute or chronic. The earliest case of true abscess was seen four days after the time of onset of symptoms. This patient showed remarkable improvement upon removal of a foreign body and bronchoscopic drainage. He was discharged as cured, and has remained so up to the present time. In the majority of

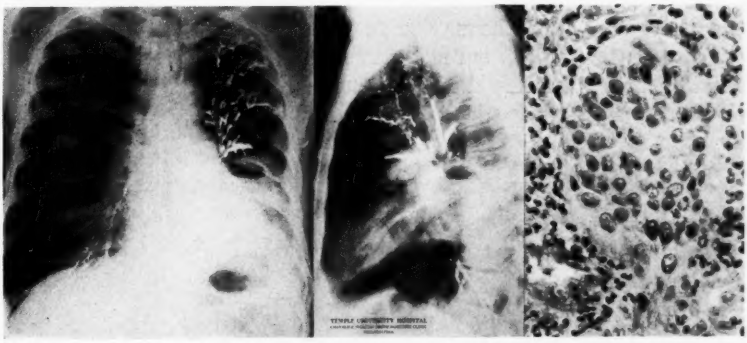

Fig. 2.—The value of diagnostic bronchoscopy was illustrated by this case. Although the patient was referred for treatment of a pulmonary abscess, bronchoscopy revealed a bronchial carcinoma and permitted removal of tissue for biopsy. Lipiodol bronchography demonstrated bronchial obstruction but did not show its nature.

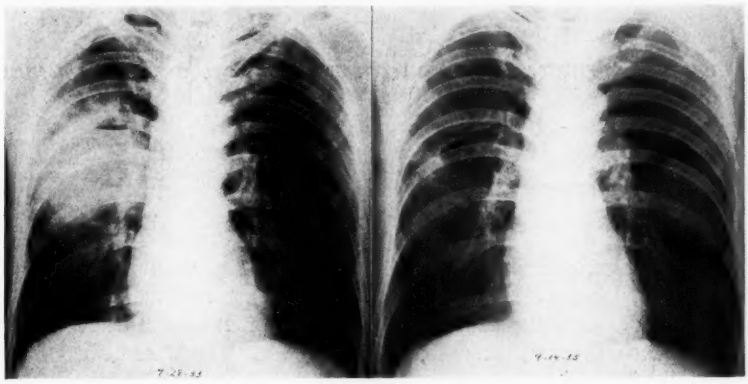

Fig. 3.—Case of pulmonary abscess of one month's duration in a man 44 years of age. The etiology was indefinite, but according to the patient, he "caught cold." Response to conservative treatment was very satisfactory, resulting in a complete cure in about six weeks. The film to the left shows the lesion at the beginning of treatment, and the film to the right shows complete clearing. Seven bronchoscopic aspirations were carried out in this case.

cases, however, symptoms have existed for a considerable period. The average is about eleven months, the shortest period being four days, and the longest, eleven years. This fact alone, we believe, has a direct bearing upon the indications and ultimate outcome of bronchoscopic or other treatment. The longer this particular type of suppurative process has

been present, the higher will be the incidence of complications, resulting in higher morbidity and death rates. An abscess wall will have been formed, fibrosis will have occurred, and associated bronchiectasis will have developed. Continued absorption of toxic by-products has manifested itself in the degeneration of parenchymatous tissue, notably the heart muscle and renal substance. These cases frequently cannot be cured without severe major surgical procedures, frequently lobectomy or pneumonectomy. But because of the pulmonary or physiologic state of the individual patient, he may be unable to withstand major surgery; in which case, the outlook is for a varying period of actual or partial invalidism and premature death. Bronchoscopy can and does offer considerable palliation to these patients, but cannot be expected to be curative.

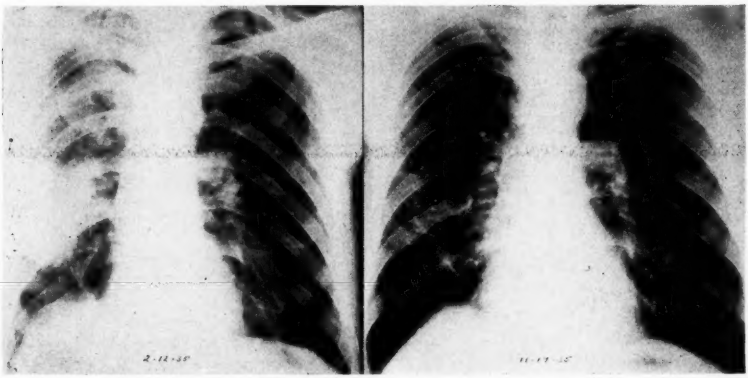

Fig. 4.—Pulmonary abscess following a cholecystectomy under ether anesthesia. Complete clearing took place in about two months under conservative treatment, as shown by film to the right. This patient received, as a part of his treatment, nine bronchoscopic aspirations.

The period over which conservative treatment can be employed satisfactorily must be individualized definitely. In this, as in so many other conditions, the problem is one of early diagnosis and early treatment. Then, and apparently only then, will there be witnessed a decided drop in the morbidity and ultimate mortality rates. The place of bronchoscopy may be viewed according to definite, but not clearly demarcated, phases in the developmental history of an abscess in any given patient. We believe, therefore, that careful consideration should be given the indications for bronchoscopic measures in relation to the chronology of the particular process. The phases, suggested by our experience, might be considered under the following headings: (1) prophylactic stage; (2) diagnostic stage; (3) acute stage; (4) subacute and chronic stages; (5) pre- and postoperative stages.

Patients treated in the first, or prophylactic, stage will not be considered in this communication, but on previous occasions Jackson and

others[4, 5, 10, 12, 13] have presented ample material to illustrate the value of bronchoscopic treatment alone as a means of removing obstruction and relieving pulmonary atelectasis, thus restoring aeration of the lung tissue and thereby preventing the development of suppurative disease which would progress into abscess formation.

Under the second heading, it may be said that there is no doubt of the value of bronchoscopy as a means to diagnosis, because this fact is now thoroughly realized and accepted. In our experience acid-fast bacilli and other evidences of tuberculosis have not uncommonly been discovered by bronchoscopy or upon the examination of bronchoscopically collected secretions when, otherwise, the diagnosis would have remained in doubt. Similarly, one patient referred to this clinic purely for the treatment of "lung abscess" was found to have a primary carcinoma of the bronchus, the abscess being secondary to the tumor.

Third, in the acute stage of pulmonary abscess bronchoscopy may initiate or provide adequate drainage, permitting the abscess to go on to spontaneous closure and healing. It is in this stage, however, that the patient must be watched carefully, so that if improvement ceases, other measures may be resorted to without delay and serious complications such as gangrene, extensive progressive suppurative pneumonitis, and bronchiectasis prevented. This period extends roughly to about four to six weeks.

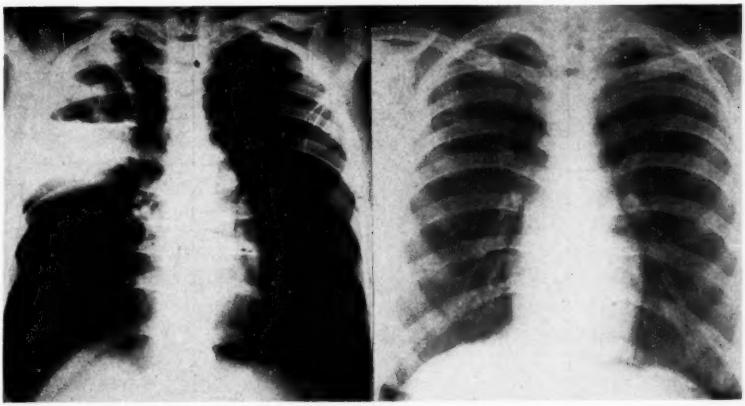

Fig. 5.—Pulmonary abscess of two years' duration in a woman 20 years of age. The film to the left shows the condition at the time of the addition of bronchoscopic aspiration to the treatment. External drainage had been contemplated but was decided against because of rapid clearing. The film to the right shows a normal chest six months later.

Likewise, in the case of an abscess that has become subacute or chronic, we believe that the conservative regime should be given a trial. If definite progress toward healing of the abscess is not soon noted, other methods of attack should be adopted. This course seems justified by the fact that in our series a number (Table IIIA) of patients in this

TABLE IIIA

RELATIONSHIP BETWEEN ULTIMATE RESULTS AND THE DURATION OF SYMPTOMS

	WEEKS			MONTHS											YEARS						INDEFINITE	TOTALS
	1-2	2-3	3-4	1-2	2-3	3-4	4-5	5-6	6-7	7-8	8-9	9-10	10-11	11-12	1-2	2-3	3-4	4-5	5-6	6+		
Cured	3	1	2	8	3		2		5	2				1	3	1				1	1	33
Improved			2	4	1	1	1		2	1				1	2	2						17
Diagnostic			1	1				1							1						1	5
No change, or worse				2				1								1						4
Abscess cured but residual bronchiectasis				1			1							1	1	1						5
Dead	1		2	1	2	4		1	1	2					1	2	1		2	1	2	23

TABLE IIIB

RELATION BETWEEN ULTIMATE RESULT AND DURATION OF CONSERVATIVE THERAPY IN PATIENTS NOT TREATED SURGICALLY

	0-7 DAYS	WEEKS			MONTHS										YEARS						ELSEWHERE AND HERE	TOTALS
		1-2	2-3	3-4	1-2	2-3	3-4	4-5	5-6	6-7	7-8	8-9	9-10	10-11	11-12	1-2	2-3	3-4	4-5	5-6	6+		
Cured		5	6	1	7	2	4						1		1	2	0	1	1	1		1	33
Improved	1	3	1		4		2				2	1			1							2	17
Diagnostic	5																						5
No change, or worse			2											1								1	4
Abscess cured, but residual bronchiectasis					1											1	1				2		5
Dead	1	5	1		4	4			1		1			1	1	4							23

class have responded satisfactorily to the conservative regime including bronchoscopy, with ultimate healing of the abscess. However, it will be noted in Table IIIB that after the third month those who were not cured outnumber those who were cured. It seems logical, therefore, to conclude that so-called "radicalism" is in reality conservatism in this group.

Last, the pre- and postoperative use of the bronchoscope is of proved value. All patients are bronchoscoped and thoroughly aspirated immediately preceding operation. In fact, the use of the bronchoscope during the actual operation is a fairly frequent practice, and in some instances has proved a life-saving measure. Postoperatively, bronchoscopic checkup, alone or in conjunction with careful lipiodol bron chography, has been invaluable. These studies afford the surgeon or clinician an accurate means of determining the existence or nonexistence of a secondary bronchiectasis or residual abscess cavity. Bronchoscopy also affords a method whereby a patient may be carried through the infective stage of a moderate limited bronchiectasis, and thus spared further major surgery.

The conservative treatment as applied to patients seen in this clinic comprises a number of different but highly related procedures, any or all of which may be employed in the treatment of any individual patient. These practices are: (1) routine supportive care including bed rest, nursing, dietary and symptomatic treatment; (2) postural drainage; (3) bronchoscopic procedures; (4) transfusions; (5) vaccines and intravenous therapy, neoarsphenamine, etc.

It is obvious that there will be considerable variation between patients in the use of these measures, the extent of their use depending upon the patient's individual demands and somewhat upon the clinical judgment of the attending physician. Drainage of the abscess cavity by other than surgical methods is accomplished by bronchoscopic and postural drainage. In our experience, postural drainage is so seldom employed as a single procedure that it is impossible to evaluate it independently; it seems best rather to evaluate "nonsurgical drainage," which combines both postural and bronchoscopic maneuvers. As for bronchoscopy, we likewise do not believe that it alone constitutes a treatment of "lung abscess." However, it has proved a valuable procedure in many cases, and its value lies not only in the act of aspiration, but in a variety of effects. Granulations, foreign bodies, and secretions can be removed, and medications may be applied or instilled. Thus, spontaneous expulsion of retained secretions is favored. The results obtained are in some cases delayed, whereas in other cases they are quite prompt and dramatic, a complete closure of cavity resulting after one or two bronchoscopic treatments.

CONSERVATIVE THERAPY

The underlying principles of the medical or conservative regime have already been outlined. These principles include rest and supportive

care, transfusions, vaccines, intravenous chemotherapy, postural drainage, and bronchoscopic aspiration; also, consultations between the medical, surgical, and bronchologic departments. For convenience in obtaining the latter, all cases are presented and discussed at the weekly "Chest Conference."

Tables IIIA and IIIB show the summary of 87 (70 per cent) cases treated by the conservative regime alone. Thirty-two cases are believed to be cured. There are five cases in which abscesses are at present cured, but in which there is a definite residual bronchiectasis as determined by lipiodol bronchography. These patients have been under the supervision of this clinic or of their personal physicians for one year or more, and probably will be subject to acute exacerbations and remissions for the remainder of their lives. Three patients were received in a moribund condition, and nine were under the supervision of this hospital for so short a time that no classification could be made.

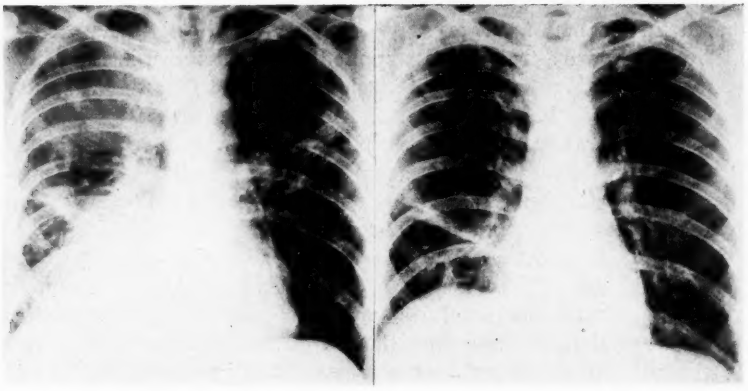

Fig. 6.—Case of pulmonary abscess and suppurative pneumonitis following tonsillectomy under local anesthesia. Complete recovery resulted in approximately four months. The patient was subjected to an intensive, well-balanced regime of conservative therapy, including fifteen bronchoscopic aspirations. The linear and annular markings noted in the film to the right are believed to represent scarring, as the patient at that time was symptom-free and has remained so until the present time.

If the "cured" and "improved" cases be considered as a group, it will be noted from Table IIIB that thirty-seven patients of a total of fifty were cured or improved within the first three or four months of treatment. It appears that the earlier the patient is seen after onset, the better is his chance for recovery either on the medical or surgical regime. However, in Table IIIB it will be observed that the death rate during those first three or four months is likewise high. The high rate is chiefly the result of pulmonary gangrene and massive hemorrhage (nine cases), both of which are the result of rapidly extending infection and occurred after a failure to improve on the more conservative regime, or after an exacerbation of an infection that for a while had

been showing satisfactory improvement. This incidentally constitutes one of the definite indications for immediate surgical intervention. For, when a patient does not show progressive improvement, or when the patient once improving regresses and "loses ground," immediate surgical intervention is imperative. On the other hand, there are a

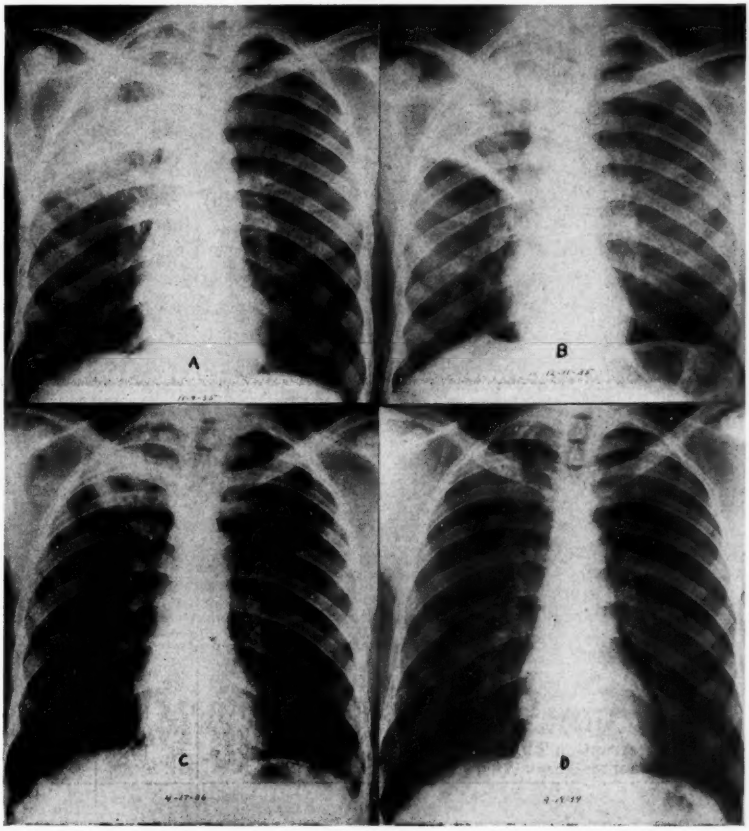

Fig. 7.—Pulmonary abscess of traumatic origin (bullet wound) in a negro male 26 years of age. *B* was made about one month after *A*, and *C* about four months later. Conservative treatment, including bronchoscopic measures, resulted in complete cure, as shown by *D*, taken approximately two and one-half years after *C*.

few patients in our series who first improved and then regressed for a time before going on to final healing. This number is small, and as in certain other acute conditions, should probably be sacrificed for the better ultimate result in the majority of cases.

SURGICAL THERAPY

The solution of the problem of pulmonary abscess lies neither in the medical regime nor in the surgical regime alone, but in the careful

coordination of both. The internist and the surgeon should be mutual consultants in each case of pulmonary abscess. Samson[26] states that "pulmonary suppuration always may be considered potentially surgical." So it is, and we are also in agreement with Samson's further statement that "the clinical course in pulmonary abscess is extremely unpredictable" and "the entire picture may change completely in a few days' time." Much of this change may be traceable directly to the adequacy or inadequacy of existing drainage. Adequate drainage, whether obtained by bronchoscopic, surgical, or other means, is an absolute necessity if the patient is to become asymptomatic and able to heal his lesion. As already pointed out, the time element is highly important if the bronchoscope is to be accorded an opportunity to be of greatest efficiency. Once the lesion becomes chronic, bronchoscopic aspiration coupled with other "conservative" measures is probably less often capable of obtaining a permanent cure. Under such circumstances, when an abscess does not continue to progress favorably, surgical intervention becomes advisable, although a period of preparation under the medical regime may render the patient better able to undergo operation. The prolongation of this period past the optimum point is not justifiable. We have observed that many times there is a period of symptomatic improvement, as well as actual improvement in the underlying pathologic condition, but if such improvement does not continue and if there is the least evidence of "lost ground," open external drainage is the procedure of choice and should be recommended without delay.

TABLE IV

RESULTS IN CASES TREATED SURGICALLY

	TOTAL	CURED	IMPROVED	DEAD
External drainage	20	13	2	5
Lobectomy	4	1	0	3
Pneumonectomy	2	0	0	2
Drainage of empyema and abscess	4	1	1	2
Thoracoplasty	3	0	0	3
Phrenic interruption	3	0	1	2
Artificial pneumothorax	1	0	0	1
	37	15 (40.5%)	4	18 (48.6%)

During the earlier years of the present survey, certain procedures were undertaken that have since been virtually abandoned as initial or combined methods. In the light of present knowledge, phrenic nerve interruption, artificial pneumothorax, and thoracoplasty are no longer considered advisable for uncomplicated abscess of the lung. Table IV shows the distribution of surgical cases with respect to the ultimate results. It will be noted that over the entire period of ten years there was a total of eighteen deaths. Only five of these eighteen cases occurred among the twenty cases treated by external drainage. It is only just to say that within the past five years it is found that the operative

mortality rate is almost negligible in good and fair operative risks. The distribution of the deaths among those subjected to external drainage is interesting. One patient died of pulmonary gangrene, one was a diabetic, and one died of cardiac insufficiency two months postoperatively. Two more are considered to have died as a result of extensive pulmonary suppuration and pneumonitis.

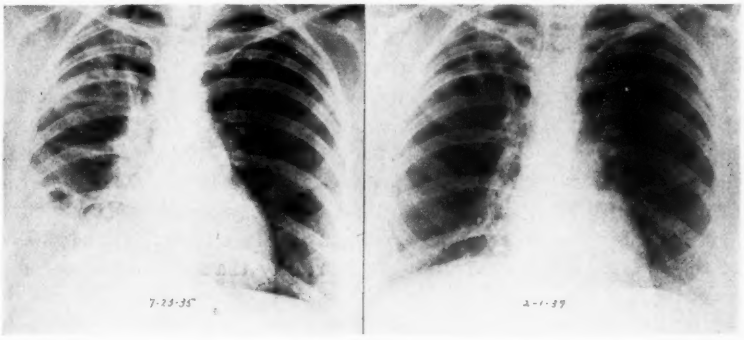

Fig. 8.—Pulmonary abscess and pneumonitis in a woman 26 years of age, of rather indefinite onset, but attributed to the patient's having "caught cold." As a result of continued conservative treatment, including bronchoscopic aspirations, a complete cure was ultimately obtained. A checkup film taken several years after discharge showed nothing abnormal but a few linear markings and slight pleural thickening.

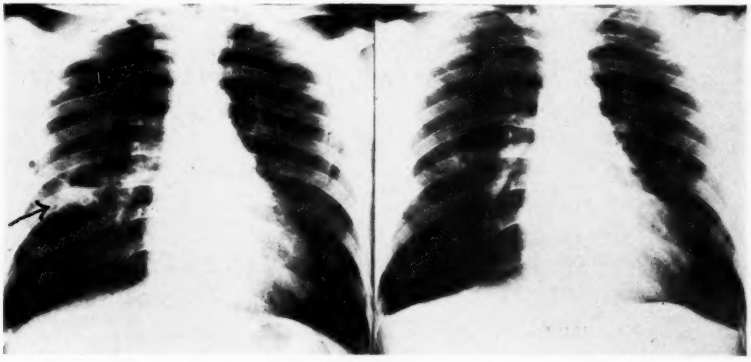

Fig. 9.—Single pulmonary abscess following a "cold" in a negro male 31 years of age. The film to the right shows complete healing of the abscess two and one-half months after the institution of conservative therapy, including bronchoscopic aspiration.

Failure of surgical drainage, like failure of "conservative" methods, is all too often the result of misapplication of method rather than the intrinsic weakness of the method itself. In any group of cases treated by any primary method, a certain number of undesirable ones must necessarily creep in, but constant vigilance will keep this number reduced to a minimum. This proper selection of cases and a realization of the importance of active special treatment with an absolute mini-

mum of delay will undoubtedly lead to lowering of the ultimate mortality and morbidity rates.

In our series of patients treated surgically, thirty-seven in all have come to operation of some sort. Artificial pneumothorax was used once, with one death. Phrenic nerve interruption was used as a primary procedure in three cases, with two deaths; the patient who was discharged to care of his personal physician has improved; his present condition is not ascertainable. There were three treated by thoracoplasty, and all three have died. Lobectomy, pneumonectomy, and drainage of abscess with concomitant empyema have been employed in four, two, and four cases, respectively, with three, two, and two deaths, respectively. In other words, there are but four survivors, two known to be cured, and two improved, among those cases not subjected to external surgical drainage. However, out of justice to the last three methods mentioned, it is well to note that they are used as primary methods more frequently in the presence of severe complications which, in themselves, carry even a higher mortality than uncomplicated abscess itself.

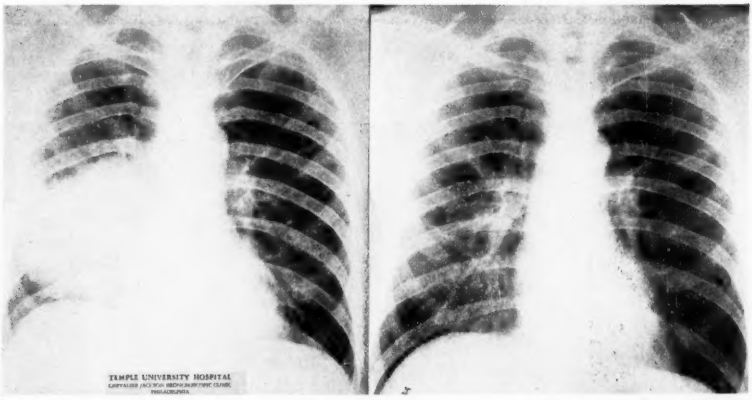

Fig. 10.—Chronic pulmonary abscess (two years' duration) following tonsillectomy under ether anesthesia in a male 35 years of age. This lesion had failed to respond to ordinary medical conservative therapy, but the addition of bronchoscopic measures resulted in complete healing within three months, as shown by the film on the right.

Surgical intervention is not to be reserved, we feel, for undesirable cases or as a secondary procedure upon patients "already doomed before operation"; on the contrary, surgical and medical programs are so interlocked that the care of abscess of the lung, whether acute or chronic, demands "the closest relationship between physician and surgeon, first rate diagnosis, accurate localization, keen judgment, and a high degree of surgical skill" (Heuer[9]).

CAUSES OF DEATH

The analysis of the causes of death in the present group presents an interesting study. Through the entire series of cases there have been a

total of forty-one deaths (33.4 per cent) up to the present time. Table V presents the complete analysis of these fatal cases. Of the causes of death listed, we feel that the most significant ones are diabetes, pulmonary gangrene, massive pulmonary hemorrhage, complicating empyema, and extensive pneumonitis.

TABLE V

LOCATION OF CAVITIES IN RIGHT AND LEFT LUNGS, RESPECTIVELY, IN RELATION TO CAUSES OF DEATH

LUNG	LOBE	LOCATION IN ALL CASES	EXTENSIVE PULMONARY SUPPURATION AND PNEUMONITIS	CARCINOMA	DIABETES	ACUTE CARDIAC FAILURE (INCLUDING POSTOPERATIVE DEATHS)	MASSIVE PULMONARY HEMORRHAGE	BRAIN ABSCESS AND EMBOLISM	BLOOD STREAM INFECTION	PULMONARY GANGRENE
Right	Upper	39	4	0	1	2	2	1	0	0
	Middle	17	3	1	0	2	2	1	0	0
	Lower	37 (15 in apex)	5	0	1	2	0	0	0	3
Left	Upper	9	0	0	0	0	0	1	1	1
	Lower	22 (7 in apex)	1	1	1	2	0	1	0	1

TABLE VI

ULTIMATE RESULTS IN 115 TREATED CASES

TREATMENT	TOTAL	CURED	IMPROVED	DIED
Surgical	37	14 (33%)	5	18 (48%)
Nonsurgical	78	33 (42%)	22	23 (29%)
	115	47 (40%)	27	41 (36%)

Although this series is not large, we note that all the patients with diabetes as an underlying complication have died. Two died without having any combined form of treatment, and one died a few hours following external drainage of the abscess. It is known that diabetes mellitus may be controlled adequately in the presence of smaller infectious lesions, but where so severe an episode as a pulmonary abscess occurs, one seems justified in assuming the situation to be grave, particularly if surgical intervention is planned. Might it not be advisable to perform an open drainage at the earliest possible moment and attempt to avoid the crisis that results from continued infection in the presence of such a systemic condition?

Extensive pulmonary suppuration and pulmonary hemorrhage (massive) are more or less a part of the same process and are the direct outcome of either chronically or acutely progressive intrapulmonary infection. In our series, the majority of deaths due to this cause have occurred after a period of neglect of the underlying pulmonary condition. We emphasize again that the longer such patients are carried along after "they have failed to improve" on the conservative regime, the more will this termination be favored. If death does not occur, at least there may be extensive pulmonary fibrosis with ensuing secondary bronchiectasis and all its related sequelae.

Putrid empyema resulting from pulmonary abscess is considered a grave complication by all concerned with pulmonary suppurative processes. When encountered with abscess of the lung, empyema is the result of rupture of an underlying abscess into the pleural cavity, or severe pneumonitis. Immediate wide and adequate dependent surgical drainage is the only advisable procedure. Even then, the mortality rate will be high. The better part of judgment seems to be to anticipate such a possibility where bronchial drainage is insufficient. The condition should be considered as an emergency and external drainage done before rupture into the pleura has occurred.

Pulmonary gangrene has resulted in death in every instance. It too impresses us as an extreme surgical emergency. The condition seems to be always fulminating and fatal. Gangrene of any other internal organ is considered the subject of immediate and grave concern, calling for emergency treatment of a surgical nature; and why not gangrene of the lung? The opinion expressed in conference, by our thoracic surgeon, W. E. Burnett, is that such lungs or lobes should be removed immediately by mass ligation if not otherwise. This seems, we believe, to warrant serious thought. Such action may prove life-saving, or at least it may lead to further knowledge that will be life-saving in the management of a condition which otherwise proves fatal.

Other causes of death do not differ from those generally encountered in the presence of chronic infection and major surgical procedures. Although they may be reduced with early diagnosis and appropriate treatment, they do not require further discussion.

DISCUSSION

There has been presented a series of 124 cases selected according to definite standards of diagnosis as outlined. All doubtful cases have been discarded. These cases have been analyzed from various angles. Of the 124 cases, eighty-three are males and forty-one are females. The reason for the inequality of numbers is not known. The cavities seem to show a predilection for the right side and for the lobar apex when occurring in the right lower lobe. This is also the experience of Myers and Blades.[21] Although ninety-three patients had abscesses in

the right lung and thirty-one in the left lung (twenty-two left lower and nine left upper), there does not seem to be any definite relationship between the location of the cavity and the ability of the patient to survive. (Table V.)

The conservative regime alone was carried out in eighty-seven cases (70 per cent), although, as stated above, nine patients were under observation for so short a time that they can scarcely be included in final statistics. In the seventy-eight remaining cases there were twenty-three deaths (29.4 per cent). Thirty-three were known to be cured; five were discharged cured, but without follow-up within the past year; seventeen improved at the time of discharge back to their referring physicians.

Thirty-seven cases (30 per cent) were referred for some form of surgical care, and of these cases, eighteen (48.6 per cent) have died of various causes (Table IV); of nineteen surviving patients, only four have survived procedures other than external drainage. It is felt that the poor results obtained by procedures other than external drainage coincide with the experience of other investigators and justify abandonment of them for the apparent operation of choice, external drainage, except where complicating conditions demand lobectomy or pneumonectomy.

The question of the optimum time for surgical intervention has been debated extensively. Neuhof[22-25] feels that abscess of the lung should not be treated differently than abscess elsewhere, and urges early operative intervention if progress is not rapid and unmistakable. The same opinion seems to be held by Bohrer.[2, 3]

Our experience leads us to agree, in general, with these opinions. However, we believe that, with few exceptions, every patient should be given the advantage of a rigid conservative regime for at least a brief period. The time limit for each patient varies widely, and the optimum time for the various therapeutic procedures can be set best by the internist, thoracic surgeon, radiologist, and bronchologist all cooperating as one, as in some sort of "chest conference."

CONCLUSIONS

A review of 124 cases of nontuberculous pulmonary abscess has been presented.

The incidence of abscess of the lung was found to be twice as frequent in males as in females.

The greatest benefit from conservative treatment is realized in the earlier months.

Patients not showing satisfactory progress and those who show signs of regressing after first improving should have prompt external surgical drainage.

Bronchoscopic measures are considered to play an indispensable role in diagnosis and to constitute an integral part of both the nonsurgical

and surgical treatment of pulmonary abscess. One should think not of bronchoscopy versus surgery, but of bronchoscopy and surgery versus the disease.

REFERENCES

1. Bloch, André, and Soulas, A.: La Bronchoscopie, État Actuel de la Question, Monographie, Paris, 1931.
2. Bohrer, J. V.: Lobectomy for Central Abscess of the Lobe, Hinge Flap for Multiple Bronchopleural Fistulas; Post-Tonsillectomy Peripheral Lung Abscess, Am. J. Surg. **34**: 83, 1936.
3. Bohrer, J. V.: Post-Operative Lung Abscess, Am. J. Surg. **35**: 64, 1937.
4. Clerf, Louis H.: Pulmonary Abscess Following Tonsillectomy, Arch. Otolaryng. **11**: 192, 1930.
5. Clerf, Louis H.: Pulmonary Suppuration, West Virginia M. J. **34**: 1, 1938.
6. Furstenberg, A. C., and Hickey, P. M.: Demonstration of Lower Air Passages by Intratracheal Injection of Lipiodol, Tr. Am. Laryngol., Rhin. & Otol. Soc., p. 4, 1936.
7. Hick, J. B., Clerf, L. H., Funk, E. H., and Farrell, J. T., Jr.: Pulmonary Abscess, An Analysis of One Hundred Seventy-Two Cases, Arch. Surg. **19**: 1292, 1929.
8. Goldman, I. B.: Bronchoscope Studies, Arch. Otolaryng. **23**: 414, 1936.
9. Heuer, G. T.: The Treatment of the Acute or Recent Pulmonary Abscess, Surg., Gynec. & Obst. **70**: 472, 1940.
10. Jackson, Chevalier: Suppurative Disease of the Lung, Report on a Series of Bronchoscopic Findings, Proc. Royal Soc. Med. **24**: 1, 1930.
11. Jackson, Chevalier L., and Bonnier, M.: Technic of Bronchography, Ann. Otol. Rhin. & Laryngol. **46**: 771, 1937.
12. Jackson, Chevalier, and Jackson, Chevalier L.: Peroral Pulmonary Drainage, Am. J. M. Sc. **186**: 849, 1933.
13. Jackson, C., and Jackson, C. L.: Bronchoscopic Observations on Post-Operative Pulmonary Complications, Ann. Surg. **17**: 516, 1933.
14. Joannides, M.: Mechanics of Abscess, Gangrene and Bronchiectasis With Suggested Methods of Treatment, J. THORACIC SURG. **8**: 427, 1939.
15. Léon-Kindberg, M., and Soulas, A.: La méthode bronchoscopique dans le traitement des abcès du poumon, Presse méd. **38**: 1591, 1930.
16. Kernan, J. D.: Abscess of the Lung Relieved by Bronchoscopy, Arch. Surg. **16**: 215, 1928.
17. Lee, Walter Estell, and Tucker, Gabriel: Post-Operative Pulmonary Atelectasis, Atlantic M. J. **31**: 284, 1928.
18. Léon-Kindberg, M., and Soulas, A.: Les méthodes bronchoscopiques dans le traitement des Suppurations pulmonaires, XXII Congrès Français de Médecine.
19. May, Etienne, and Soulas, A.: Bronchoscopy in Therapy of Chronic Abscess, Recovery in Some Cases, Bull. et mém. Soc. de méd. de Paris **139**: 37, 1935.
20. Mosher, Harris P.: False Lung Abscess and Lipiodol, Laryngoscope **37**: 138, 1927.
21. Myers, Dan, and Blades, Brian: The Clinical and Roentgenologic Features of Pulmonary Abscess Located in the Superior Division of the Lower Lobes, J. THORACIC SURG. **8**: 321, 1939.
22. Neuhof, H., and Touroff, A.: Acute Putrid Abscess of the Lung, Analysis of 45 Consecutive Cases, Surg., Gynec. & Obst. **66**: 836, 1938.
23. Neuhof, H., and Touroff, A.: Acute Putrid Abscess of the Lung, Principles and Operative Therapy, Surg., Gynec. & Obst. **63**: 353, 1936.
24. Neuhof, H., and Touroff, A.: Acute Aerobic (Nonputrid) Abscess of the Lung, Surgery **4**: 728, 1938.
25. Neuhof, H., and Touroff, A.: Acute Putrid Abscess of the Lung, J. THORACIC SURG. **9**: 439, 1940.
26. Samson, Paul C.: Bronchoscopy in Relation to Thoracic Surgery, West. J. Surg. **47**: 687, 1939.
27. Soulas, A.: Bronchoscopic Therapy of Fetid or Putrid Abscess, Presse méd. **44**: 291, 1936.
28. Soulas, A.: Bronchoscopo-Therapy in Bronchopulmonary Suppuration: Its Mechanism and Results, J. Laryng. & Otol. **52**: 1937.
29. Soulas, A.: The Role of Bronchoscopy in Surgical Therapy, Bronchoscop., oesophagoscop. et gastroscop., pp. 223-228, Oct., 1938.

DISCUSSION

DR. PAUL C. SAMSON, Oakland, Calif.—I am always happy to discuss a paper from the Jackson Clinic. Coming, as it does, from a group doing highly specialized work, I think it is particularly refreshing to hear their repeated statement that bronchoscopy is not a cure-all for lung abscess. In my position—that is, doing both bronchoscopy and thoracic surgery—I may be able to stress several points that I believe need repeated emphasis.

Unfortunately, the statistical tables show what seems to be the average situation today as far as lung abscess is concerned, namely, that pulmonary abscess, with the exception of malignancy, is still the most fatal common disease with which we have to deal in the chest. When comparative figures are listed for other conditions, the relative mortality rate in abscess is appalling.

There are several remedies for this. One of the most important is education of the general medical group as to the danger of abscess and danger of delay in adequate treatment. I believe that we, as an association composed of surgeons, internists, and roentgenologists, have a definite part in this campaign as far as the general medical public is concerned. The very fact that the average length of time in the Jackson series before the patient was seen was eleven months is, I think, confirmatory of this fact.

As to treatment, there are only two words essential—*adequate drainage*, however it is obtained. The question of whether this is happening in any particular case should be solved by the close cooperation of the radiologist, surgeon, and internist. It is high time we stopped talking about the distinction between medical and surgical treatment. Such a course leads to error and disaster in this condition. Pulmonary abscess is extremely unpredictable, and indication for change of therapy comes often in the course of hours or days, rather than in the course of weeks or months. For this reason, early consultation between internist, bronchoscopist, and surgical consultant should be carried out. Bronchoscopy is in the picture, both in diagnosis and therapy. Disclosure of undiagnosed tumors and undiagnosed foreign bodies is probably most important. It may be one additional means to promote adequate drainage and cure.

All of us have had the following experience too often to think it is merely coincidental. A patient is on ordinary conservative therapy with bed rest, postural drainage, etc., getting along about so well; bronchoscopy is added and there is immediate improvement.

To summarize, my own personal feeling about abscess is somewhat as follows: Every abscess, with the exception of those with severe hemorrhage or showing brain involvement, is entitled to at least one bronchoscopy. I do not even except patients who are apparently moribund, because often this may be due to impending suffocation from vomica. Certainly they are entitled to bronchoscopic aspiration, and that is the only thing in such a case which will save their lives. In the vast majority of cases, it can be told within a relatively few days following the first bronchoscopy whether or not bronchoscopy will be to their advantage. If the improvement is questionable, another bronchoscopy may be tried. If there is no improvement, external drainage should be considered within a few days.

In general, we feel that the more chronic the abscess, the less chance there is for bronchoscopy to have an effect, and it is my own clinical impression, which I cannot back up with bacteriologic study, that the more putrid an abscess, merely by the gross smell, if you will, of the secretions, the less chance bronchoscopy has of being successful in promoting adequate drainage.

In conclusion, bronchoscopy is an invaluable procedure diagnostically and therapeutically. Let us use it early. Let us get our friends in the general medical profession to permit us to see the abscess cases early, and let us not continue on

any course for more than a few days, or at most for a week or two, if it seems that course is not bringing progressive and certain cure. Thank you.

DR. W. E. BURNETT, Philadelphia.—It has been my good or ill fortune to be associated on several of these cases that Dr. Judd and Dr. Jackson are reporting, and although the time is short, there are one or two items I thought might be worth discussing in an effort to reduce the mortality.

(Slide.) This is an example of a girl of 21 years, who is representative of some of the earlier cases. She had been sick for six months, and, as you see, almost the entire right upper lobe had cavitated. This patient was drained after about five months of bronchoscopic treatment, but developed a fatal brain abscess from an embolus. Subsequently on two occasions we have drained such lesions, allowing the infection to subside, and then have treated them by lobectomy. Those two patients recovered very nicely.

(Slide.) There is another item, which has nothing to do with the mortality, to which I shall return in a moment. That is this next patient whose abscess was of very recent origin after a tonsillectomy; after a period of three weeks' bronchoscopic treatment, he showed excavation but no improvement, and was turned over to us for surgical drainage. He made a very nice recovery in a period of two months, becoming symptom-free and showing little or no scarring radiographically. What appears to be cavity is rib regeneration about the drainage wound. However, it is an example of what happens in many cases, even in some of those that we drain very early. Although they are clinically symptom-free, there is evidence on bronchography of a small- or fair-sized cavity, which apparently is well epithelialized, well drained, and producing no symptoms. We have noticed this in several patients, both those with delayed and with early drainage.

(Slide.) Because they are well drained, they produce very few symptoms, but they indicate definitely that there has been destruction which has not been replaced.

(Table V.) There were three patients with pulmonary gangrene, all of whom died, although the gangrene was confined to one lobe. It seems logical, by applying the principles of surgery elsewhere, as in the treatment of a gangrenous leg or gangrenous appendix, or possibly a gangrenous gall bladder, although that is controversial, to feel it might be wise to amputate such lobes by mass ligature and get rid of the source of absorption. I believe this is a justifiable thought, although we have not tried it deliberately, except in one case of an attempted Rienhoff two-stage method of pneumonectomy. On the second day the patient's temperature rose from normal to 102° F., and on the third day to 104° F.; although thoracoscopically the lung was still shining and apparently living, we performed the second stage that afternoon, with the result that the temperature dropped to normal and stayed there. Although the patient eventually developed an empyema and died of it, because it was not recognized, it indicated how quickly the response would occur to amputation of a necrotic portion of the lung. I think it is worth deliberate trial if gangrene can be recognized. Thank you.

DR. JACKSON.—I would like to thank the discussors, particularly Dr. Samson, and also to express my regret that the lateness of the hour apparently has made the discussion a little bit shorter than it might have been.

One question that was asked me, over my shoulder, by a man who felt it was too late to get on his feet, had reference to the permissible frequency of bronchoscopy for diagnosis or treatment. I can say in reply to that question that frequency depends almost entirely on indications. We do not hesitate to do bronchoscopies as frequently as every other day, or even every day, if there is some particular reason for them in the individual case. Patients who are receiving a systematic course of bronchoscopic drainage may have a treatment as often as once or twice a week. We modify the schedule according to progress.

With regard to the things that affect our decision as to whether or not bronchoscopy should be done on a particular day, I might say, for example, that fever may be either a contraindication or an indication for the particular bronchoscopy. If we expect to be able to produce an improvement in drainage at once by some manipulation, the elevation of temperature may be a reason to do the bronchoscopy and attempt to produce the drainage. If, on the other hand, we think the fever is due to a spreading of pneumonitis or something of that sort, we would, of course, postpone that bronchoscopy.

I would like to mention that Dr. Millard Arbuckle of St. Louis, who is associated with Dr. Graham, read a paper on Wednesday of this week at the meeting of the American Broncho-Esophagological Association in New York, in which he reported forty-three cases of pulmonary abscess, and of these forty-three cases he stated that 50 per cent had responded favorably to bronchoscopic treatment. That paper will be published in the near future. [Ann. Otol., Rhin. & Laryng., Sept., 1940.]

There is one last slide showing the tabulation of the ultimate results. I would be very grateful, Mr. Chairman, if you would give Dr. Judd just a minute to discuss this slide.

DR. JUDD.—Table VI shows a summary of the results obtained during the past ten years. We treated a total of thirty-seven cases by surgical methods. Fourteen are cured, five improved, and eighteen have died, making a total of 33 per cent cured and 48 per cent dead. Of the seventy-eight patients treated by nonsurgical methods, thirty-three are cured, twenty-two improved, and twenty-three dead, making a total of 42 per cent cured, and 29 per cent dead. In the grand totals of patients treated, 115 cases of 124, 40 per cent are cured by surgical and nonsurgical methods and 36 per cent are dead.

If we were to break these figures down into two five-year periods, I think we should find the contrast rather striking, because our conception of the indications for treatment has changed, and, likewise, the methods of treatment have changed in this second five-year period.

I would like to emphasize again what Dr. Burnett said about gangrene and the total removal of the affected part, either the lobe or the entire lung, and also to mention the fact that it has been our experience, even though the series is small, that all diabetic patients have died. Might not this also be considered a surgical emergency and not subjected to any greater delay than necessary, and therefore be operated upon by external surgical drainage as soon as the patient is seen?

II. SOME CONTROVERSIAL MATTERS OF ANESTHESIA FOR THORACIC SURGERY*

HENRY K. BEECHER, M.D. (BY INVITATION)
BOSTON, MASS.

TOO often attempts to improve a process result in making a relatively easy job hard. This unfortunately appears to be true in the case of anesthesia for thoracic surgery. Throughout the building up of a rather large series of cases† at the Massachusetts General Hospital, a prime consideration has been to discover the really essential parts of a satisfactory technique for these cases. Any factors which contribute to the safety and success of the operation are considered essential. Any factors not distinctly in this category are to be eliminated, even though not clearly bad.‡ Many innovations in this field can be ruled out on logical grounds; others must be tried out. This paper is the record of our attitude at the present time toward various practices recommended for use in anesthesia for thoracic surgery, with what we hope are logical reasons for or against the adoption of many of the practices discussed in this field and labelled indispensable at various clinics. Some continuity is given to an otherwise disjointed examination of current practices by their application or rejection in our technique.

With standardization of surgical procedure, it may be expected that more thoracic operations will be carried out by a greater number of surgeons. In some cases this may be desirable, yet as long as the anesthesia technique is pictured as possible only for an expert with wide experience and much equipment, it is not likely that even "standardized" thoracic operations will be attempted in any except the large clinics. Whether these operations should, even when standardized as much as possible, be turned over to the occasional thoracic surgeon is not a question for me to attempt to answer. Certainly any such evolution cannot occur unless it can be shown that good anesthesia can be provided for these operations by the average well-trained anesthetist who may have a wide experience in providing anesthesia for general surgery, yet very little experience in thoracic surgery. Therefore, a further aim has been to discover if we could develop a sound technique with the simple apparatus found in or easily obtainable by all hospitals. The test of our results must be that they equal or excel those obtained with more complicated techniques and apparatus.

From the Anesthesia Service of the Massachusetts General Hospital.

Read before the Twenty-Third Annual Meeting of the American Association for Thoracic Surgery at Cleveland, Ohio, June 6, 7, and 8, 1940.

*This is the second in a series of articles with the general title "Anesthesia for Thoracic Surgery." The first article was "Principles of Anesthesia for Lobectomy and Total Pneumonectomy," *Acta med. Scandinav.* **90:** 146, 1938.

†Forty-four total pneumonectomies and 172 lobectomies to April 1, 1940.

‡To simplify the problem as much as possible, conclusions have been drawn from the experience of one surgeon, Dr. E. D. Churchill, except in the thoracoplasty group.

ANESTHETIC AGENTS FOR USE IN THORACIC SURGERY
ETHER VS. CYCLOPROPANE

Oxygen Available.—Many patients who come to the thoracic surgeon have a serious limitation of vital capacity. The procedures of the thoracic surgeon often tend to reduce an already lowered vital capacity. There can be no question that the possibility of using a high percentage of oxygen with any desired depth of anesthesia is a characteristic of great value in anesthetic agents. Two agents in common use possess this qualification, ether and cyclopropane, although with ether it is possible to use about 20 per cent more oxygen than with cyclopropane at the depth of anesthesia needed for surgery around the hilum. This, coupled with the suspicion (cf. C. A. Smith[8]) that cyclopropane may interfere with the oxygen uptake of cells, puts ether in the position of being at least as good as and perhaps better than cyclopropane in this respect.

Circulatory Strain.—The strain on the circulatory system is considerably increased when the pleura is open to atmospheric pressure. The ideal anesthetic agent should not increase this. The tolerance of patients with frank circulatory impairment for ether is well established. It has also been well demonstrated that cyclopropane has a toxic effect on the heart in a relatively high percentage of patients. Whether toxic cardiac effects are responsible or not, the mortality rate appears to be about twice as high under cyclopropane as under ether. This comparison is made on the basis of experts' figures for cyclopropane against data from less skilled sources for ether, a comparison which should give the advantage to cyclopropane, but does not.

The final test in this regard must be made in the clinic. Although cyclopropane was used at the Massachusetts General Hospital for more than four years, we found it to be the rule, in a small number of thoracic cases, that about an hour after induction, the blood pressure began to fall in these cases, whereas under ether, this "fatigue" usually did not appear until three hours or more of anesthesia and surgery. A rather high incidence of low blood pressure following thoracic surgery under cyclopropane has also been observed and reported by Rovenstine (1935).[7]

Irritation.—Ether undoubtedly is more irritating to the mucous membranes than cyclopropane, although the latter agent is not without irritation, as evidenced, for example, by salivation and occasional laryngeal spasm. Salivation and the flow of mucus stop during full surgical anesthesia, so this offers a problem only during induction and recovery. The reported accumulation of mucus in the bronchi and trachea during full surgical anesthesia is believed to be due to accumulation there by drainage from smaller bronchi where it had been deposited during induction. Facilities for bronchial aspiration are essential, we believe, for

other reasons; so if mucus is troublesome, it can easily be aspirated. During recovery the cough reflex is present by the time the renewed secretion might cause difficulties. The cough reflex is usually active by the end of the operation under ether.

Is the irritation of ether of morbid or of mortal importance? This is a difficult question to answer. We can get information of value from various directions. More and more information is accumulating which indicates that the anesthetic agent is not responsible for postoperative pulmonary complications in general surgery: the 2 to 1 higher incidence in men than in women, the much higher incidence of complications after upper abdominal rather than after lower abdominal operations, the equal incidence after general, local, or spinal anesthesia as reported by several observers. Following many hundreds of thoracic operations under ether, we hardly ever have seen the type of postoperative pulmonary complications found after gastric surgery. A fairly wide experience with ether (about 350 anesthesias) in the presence of active pulmonary tuberculosis, with good results (to be published in detail later), offers, with the points just mentioned, strong argument that the irritating quality of ether is not important in morbidity or mortality.

NITROUS OXIDE

Nitrous oxide is of great value in inducing anesthesia in thoracic surgery when it is possible to do so without producing clinical signs of anoxia. When the oxygenating power of the body has been crippled to the extent that the stage of surgical anesthesia cannot be entered under nitrous oxide without anoxia, skillful use of the agent still permits it to be of value in diminishing the irritant effects of ether, so that patients can be induced into ether anesthesia without complaint or anoxia. Eighty per cent nitrous oxide is useful for producing analgesia in conjunction with local anesthesia. Nitrous oxide alone will not give adequate depth of anesthesia.

AVERTIN

For some years avertin was frequently used as a "basal anesthetic" for thoracic surgery at the Massachusetts General Hospital. About four years ago it became apparent, with increasing experience, that even the so-called "small basal doses" (70 or 80 mg. per kilogram) would too often produce respiratory and circulatory depression in these patients who were peculiarly liable to suffer from the results of such depression. An examination of our full experience (about 4,000 administrations) with the agent in all cases indicated that, even when the bad choices were eliminated, the death rate was of the order of 1:1,200, far too high a mortality rate. It was found that the effects of this agent were not only highly undesirable, but were also unnecessary. The use of avertin in thoracic cases was abandoned. In our opinion, the combination of avertin and nitrous oxide for use in thoracic surgery can

only be condemned. Direct reports from clinics where this combination is used support this opinion.

EVIPAL AND PENTOTHAL

The use of evipal and pentothal has become widely established, apparently justly so, for a wide variety of cases. Employment in major thoracic surgery is not one of the legitimate uses of these agents. Their toxic effects are exerted chiefly on the respiratory and circulatory systems, the very systems most vulnerable in this group of cases. These agents excel for short procedures; major operations in thoracic surgery too often take three or four hours. These two agents have been rather widely used for induction of anesthesia preceding thoracic surgery. It is true they can provide a pleasant and simple induction of general anesthesia. We believe the patient pays too high a price for this smooth induction when the respiratory and circulatory systems are to be under the strain of prolonged open chest surgery. In any case, the advantage of an induction with these agents over a properly given nitrous oxide induction is questionable. The anesthetist who is unable to provide a pleasant and safe nitrous oxide-ether induction has little business employing the barbiturate agents for the purpose.

LOCAL ANESTHESIA

Local anesthesia has established its value for use in minor thoracic operations, such as the drainage of empyema and lung abscess, thoracoscopy, etc. We believe its use to be of value in blocking the vagus nerve and its pulmonary plexus, the phrenic nerve, or the heart's surface when troublesome responses arise from stimulation of these structures during thoracic operations under general anesthesia. The use of local anesthesia as the sole anesthetic agent for major open chest surgery is open to many of the same criticisms that can be applied to spinal anesthesia for this purpose. These are discussed below.

SPINAL ANESTHESIA

In an article written some time ago, I pointed out[2] that anesthesia for open thoracic operations should be designed to overcome three hazards: obstructing secretions, troublesome reflexes, including cough, and anoxia. Under spinal anesthesia I submit that none of these three hazards can be as efficiently combated as under general anesthesia. While an intratracheal tube can be introduced under local anesthesia, difficulty may be experienced with it during spinal anesthesia in the long operations necessary in thoracic surgery. Without an intratracheal tube, the instantaneous and at times life-saving bronchial aspiration will not be possible. Positive pressure anesthesia is apt to be less efficient without an intratracheal tube than with it. The use of positive pressure in the airway of a conscious patient is poorly tolerated.

Often a considerable point has been made of the maintenance of the cough reflex in patients under local or spinal anesthesia. An effective cough requires a tight chest wall, so that a high intrathoracic pressure can be built up. This is impossible during open thoracic work or after resection of ribs has mobilized a segment of the chest wall, and therefore a cough cannot be efficient, as pointed out by Brown and Archibald.[4] Cough has a spreading as well as expulsive action (op. cit. and Ameuille[1]). When the airway contains irritating, infectious material, any attempt to cough may spread this to the lung tissue which is to remain. The anesthetist, not the patient, must accept the responsibility for maintaining a clear airway.

The occasional crippling effects of high spinal anesthesia on the respiration and circulation are well known. Such interference is extremely undesirable in these cases. The statement sometimes made, and offered as an advantage, that little bleeding occurs during thoracic surgery under high spinal anesthesia requires no comment. If respiratory failure, hemorrhage, or a fall in blood pressure occurs, it is more difficult to cope with it under spinal than under general anesthesia.

Finally, the published mortality figures for spinal anesthesia in thoracic surgery are anything but reassuring.

We do believe, however, that there is a rare though legitimate use for spinal anesthesia when a bronchial fistula is present and the cautery must be employed in malignancy involving the chest wall. Probably some lung abscesses could be operated upon properly under spinal anesthesia, provided the region for the spinal puncture was not heavily contaminated.

POSITIVE PRESSURE

Thoracic surgery has now generally reached the stage of development where it is not enough to "get away" with a given procedure. One must "get away" with it in a very high percentage of cases. I believe it has been demonstrated clearly that for success in any sizeable number of cases, positive pressure in the airway is necessary at times in some cases, and almost constantly in others. To insure effective positive pressure as desired, intratracheal intubation is important.

During open thoracic operations several desirable objects can be accomplished with certainty only by positive pressure in the airway:

1. The prevention of collapse of the contralateral lung and insurance of adequate gaseous exchange between the alveoli and the blood. Positive pressure in the airway may not be necessary for this purpose when the mediastinum is stabilized by old scar tissue; otherwise, it often is.

2. The frequent reinflation of collapsed regions of lung during operation, lung which sometimes becomes edematous (presumably from local anoxia) during operation if allowed to remain long collapsed.

3. The demonstration at intervals during operation of lines of demarcation in segmental pneumonectomy.

4. The stabilization of the mediastinum, with reduction of movement of the surgical field and avoidance of the harmful respiratory and circulatory reflexes often set up by swinging of the mediastinum. Frequently such stabilization appears to be of great importance.

5. The re-expansion of collapsed lung and reduction of pneumothorax at the time of closure of the chest wall. We believe it unwise to expand forcibly areas of lung which have been long collapsed, that is, collapsed preceding the operation. Such areas may suddenly become grossly edematous if abruptly reinflated. Gradual reinflation of such areas during the convalescence appears to be the best procedure. These areas will usually reinflate spontaneously, though gradually, as the negative intrapleural pressure increases. If the long-collapsed areas easily reinflate with the newly collapsed areas at the end of operation, they will not be likely to cause difficulty. In any case, we believe that positive pressure of 20 cm. water should not be exceeded, even briefly. Usually half of this is adequate for reinflation.

INTRATRACHEAL TUBES

The use of intratracheal tubes is desirable in surgery through the open pleura. Such use appears indispensable for a low mortality in the group of cases where pus or blood in the airway may be encountered during operation. We also believe intratracheal tubes provide an indispensable safeguard in any operation in which the pleura may be accidentally opened, as in cardiac and pericardial surgery, dorsal sympathectomy, substernal thyroidectomy, and so on. Intratracheal tubes are unnecessary, even contraindicated, in thoracoplasty for tuberculosis, unless the sputum is so profuse as to constitute a real threat to the oxygenation of the patient, in which case intratracheal tubes are used notwithstanding the possibility of new foci of infection developing in the trachea where slight trauma may have been produced by pressure of the tube on the mucosa.

Intratracheal tubes have two major functions in thoracic surgery: to insure an open airway and the effective use of positive pressure when desired; and to permit immediate and frequent bronchial aspiration when sputum or blood may obstruct the airway. Another function might be added, namely, that of abolishing cough by making closure of the glottis impossible.

During critical periods when a gush of purulent material may be thrown into the airway by manipulation of a diseased lung, or when bleeding into the airway may have occurred, the anesthetist is easily the busiest man on the team. Rapid, efficient bronchial aspiration may be of life or death importance. Patients who are under moderate cyclopropane anesthesia at such a time tend to become too light and interfere with aspiration. This is not the case with ether.

PLACEMENT

Intratracheal tubes should nearly always be introduced under direct vision through the mouth. This is always the case when the coiled wire spring tubes (Flagg) are introduced on a stylet. The rubber tubes (Magill) should not be introduced through the nose when infection is present there, in hypertension, when the bleeding time is prolonged, or when insertion is difficult. We prefer a Jackson laryngoscope, with the battery and switch remote from the airway, in order to reduce the possibility of spark in the presence of explosive gases when these tubes are placed under general anesthesia. The Jackson laryngoscope can be kept at hand and used to introduce a bronchoscope if necessary to facilitate the removal of necrotic debris or clots not removed by bronchial aspiration. Use of the bronchoscope at the end of operation may also be desirable if stethoscopic examination suggests bronchial obstruction of a segment of lung. It is always desirable to have a bronchoscopic set in the operating room during these operations.

Whenever the sputum is very profuse or massive hemorrhage is feared during induction, the intratracheal tube may be inserted under local anesthesia before starting the general; otherwise, the tube can be placed under general anesthesia.

TYPES OF INTRATRACHEAL TUBES, THEIR PROS AND CONS

Magill.—These rather soft tubes can be used when no sputum or blood is anticipated in the airway. Aspiration through these tubes may be rather slow.

Flagg.—These coiled wire spring tubes as modified by Woodbridge are best when rapid bronchial aspiration may be necessary.

Other Types of Intratracheal Tubes.—Some tubes contain coiled wire springs permanently imbedded in rubber. The tube wall is apt to be unjustifiably thick here.

Intrabronchial Tubes.—Several hazards are associated with the use of these tubes. Aside from the technical difficulties of their use and the possibilities for trauma during their insertion, they are prone to come out of place at inopportune times, even in the hands of those who are skilled in their use. This constitutes a serious hazard, for with them in place, it is difficult, if not impossible in many cases, to keep the area surrounding these tubes free from pus, necrotic material, and blood which may drain into it from the diseased lobe. If the intrabronchial tube comes out of place, the good lung may be filled suddenly with this irritating, infectious material and the airway blocked by its quantity, whereas, if this material had been aspirated efficiently as it gathered, no such result would have occurred.

Even if these tubes could be held in place, their use is still contraindicated in many cases. If it is known with certainty before operation that an entire lung is to be removed, the use of an intrabronchial

tube inserted into the good lung may be at least theoretically sound in keeping the good lung free from blood and irritating material. With this tube in place, considerable interference with aspiration of the diseased lung will be the case, with spread of irritating or infectious material throughout that lung. If the entire lung is to be removed, this spread makes no difference. If, during the course of operation, it is found to be unnecessary to remove the entire lung, any tissue which is then left in has been jeopardized, as well as the patient's life, for the inadequately aspirated, irritant, obstructing material has probably spread widely throughout the lung and it is likely that pneumonitis will develop in the remaining tissue.

Inflatable Cuffs.—The use of an inflatable cuff on the intratracheal tube is not necessary* and is unwise for several reasons. Often rather profuse drainage from the diseased lobe will trickle into the trachea at a slow rate, so that it is impossible to pick up much of it by a catheter through an intratracheal tube. In the absence of the inflatable cuff much of this slow drainage follows along the outside of the intratracheal tube and drains into the face mask, since the patient is in the head-down position.† In the presence of an inflatable cuff, a considerable quantity of this slow spillage may enter the good lung if it cannot escape alongside the intratracheal tube (Fig. 1). In Fig 1 the tube is represented in its usual position. With a much shorter tube, the hazard from slow spillage would be avoided. A very short tube, however, would introduce other hazards and would not protect against sudden gushes of purulent material.

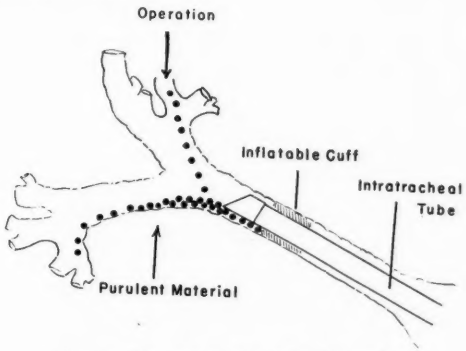

Fig. 1.—Diagram of a tube in the trachea.

When gushes of purulent material suddenly enter the airway or a profuse intrabronchial hemorrhage occurs, the speediest aspiration may not get all of it; the material cannot all enter the intratracheal tube.

*It might be expected that without an inflatable cuff around the intratracheal tube, gastric distention would result from the positive pressure employed in the airway. We have not seen this happen except in esophageal surgery through the open chest when the esophagus was being tented up by the surgeon. Here the distention was easily relieved by a gastric tube opened at intervals.

†To promote drainage and to reduce the possibility of emboli.

In the absence of an inflatable cuff, much will flow down alongside the intratracheal tube and out into the face mask, since the Flagg tube usually only half fills the trachea. In the presence of an inflatable cuff this overflow material enters the good lung. We commonly find that 3 or 4 oz. of pus and necrotic material have drained into the face mask during the course of a lobectomy for bronchiectasis, notwithstanding very frequent bronchial aspiration. Since we use the Bradshaw type of face mask with the intratracheal tube orifice outside the mask, this material does no harm.

It seems probable that inflatable cuffs sufficiently tight to hold positive pressure of 20 cm. water may damage the mucosa or its delicate ciliary apparatus, making it ineffective at least temporarily for a period following the operation.

Finally, at least one fatality has been directly due to the use of these tubes. This accident as described by Lennon and Rovenstine[6] occurred in a young man of 25 years:

> "Exactly one hour and forty-two minutes after the endotracheal airway had been introduced there was heard a muffled report like that made by discharging a toy pistol. The sound seemed to come from within the patient's chest. The noise was heard by all members of the operating team and visitors in the room.
>
> "It was immediately noted that the closed system for anesthesia was no longer leak proof. The rebreathing bag deflated rapidly although the flow of gases was not changed."

The patient went into shock. Subcutaneous emphysema developed, and the patient died twenty hours following operation. Autopsy confirmed the assumption that the trachea had been ruptured by a blowout in the inflatable cuff. The report concludes with the remark that "it may be stated that this fatal accident in no way invalidates the use of an inflatable cuff on an intratracheal airway. . . ." I cannot agree with this. Inasmuch as inflatable cuffs are unnecessary, even contraindicated in many cases, the fact that they have been demonstrated to have caused a fatality is a further argument against their use.

BRONCHIAL TAMPONAGE

Various agents, such as rubber balloons and gauze packs, have been employed to close off the diseased lobe or lung in order to interrupt drainage therefrom. The principle of their use is sound, if the irritating, obstructing material can be confined to the region to be removed. In the case of lobectomy, the firm insertion of the tampon may be technically quite difficult. Aside from technical hazards, the problem of insuring its tight fit until the proper moment for removal is considerable, for if the pack becomes dislodged and unexpectedly releases a sudden flow of material into the airway, the result is apt to be unfor-

tunate. The insertion of the pack to insure its tight fit and yet easy removal at the proper time prolongs the wear on the patient preceding the induction of general anesthesia. Unless these tampons are nearly perfect, they are distinctly bad. I have had no firsthand experience with them.

"CONTROLLED RESPIRATION"

It has been advocated repeatedly recently that respiratory failure be produced by deliberate overventilation of patients under anesthesia and that this be followed by artificial ("controlled") respiration by the anesthetist. This recommendation has been described as advisable chiefly for two reasons: first, in order that the operative field might be as quiet as possible; and second, to insure proper ventilation of the lungs, with avoidance of accumulation of carbon dioxide in the blood. For several reasons we do not believe this technique is necessary or advisable:

1. An experienced surgeon is rarely seriously disturbed by the patient's respiratory movements, even in thoracic surgery.

2. Crafoord's[5] rather alarming data obtained from experiments on dogs suggested that a dangerous piling up of carbon dioxide might occur in the blood stream of patients when the pleura was open. This has also been taught by others. From our own data obtained in patients, such does not appear to be the case. While this study is not yet complete, our failure to employ "controlled respiration" has not so far during operation caused any elevation of the arterial blood carbon dioxide in the twelve patients* examined. This study will be published later, with methods.

3. The deliberate production of respiratory failure in patients under anesthesia removes the greatest safeguard against overdosage. With the respiration eliminated, the anesthetist's first real warning of serious overdosage comes only at about the time signs of circulatory failure are established. This is particularly unfortunate in the group of cases under discussion, for once circulatory failure begins, it is sometimes extremely difficult, if not impossible, to check it.

Aside from the three cogent reasons just mentioned, we do not yet know that the alkalosis necessary for "controlled respiration" is compatible with safety in other respects in the sick, often debilitated patients who must undergo thoracic surgery. There is some evidence that this procedure may result in increased strain on the circulatory system.

SUMMARIZING DISCUSSION

Various agents and techniques have been considered for anesthesia in thoracic surgery. Our own technique has evolved over a period of several years and includes a preference for the following in open pleura operations:

*Six lobectomies, one thoracic exploration, one mediastinal tumor excision, four total pneumonectomies.

1. Ether anesthesia induced with nitrous oxide, occasionally supplemented by procaine block of the vagus nerve and its pulmonary plexus, the phrenic nerve, or the heart's surface.

2. Intratracheal intubation (Flagg tube when obstructing or irritating secretions are anticipated in the airway, avoidance of both inflatable cuffs and intrabronchial intubation).

3. Facilities for rapid bronchial aspiration, as indicated.

4. Positive pressure anesthesia when necessary.

5. Head-down position.

We believe these five points cover the physical essentials for anesthesia safety and success in this type of surgery.

For objective support we submit mortality data* which compare favorably with those from other clinics. High mortality rates do not necessarily incriminate the anesthesia. On the other hand, we do not believe it is possible to get low mortality rates in thoracic surgery with poor anesthesia. Lobectomy and pneumonectomy in the presence of bronchiectasis present difficult anesthesia problems. Here large quantities of pus, the intrabronchial bleeding, and the pleural adhesions which require long operating frequently put the anesthetist and the anesthesia to a severer test than surgery for cancer.

OPERATION	NUMBER	ALL HOSPITAL DEATHS	PERCENTAGE
Lobectomy	124	3	2.4
Total pneumonectomy	9	1	11.1

We do not consider that the anesthesia deserves any great credit for these low mortality rates; rather, our attitude is that the anesthesia could not have been very bad. We believe these data establish the fact that relatively simple anesthesia procedures are distinctly satisfactory for this difficult group of cases. The establishment of this point was a major aim of this paper.

REFERENCES

1. Ameuille, M. P.: Injection transpariétale de lipiodol dans une caverne pulmonaire. Production sous l'écran du phénomène de l'embolie bronchique, Bull. et mém. de la Soc. méd. d. hôp. de Paris **48**: 791, 1924.
2. Beecher, Henry K.: Principles of Anesthesia for Lobectomy and Total Pneumonectomy, Acta med. Scandinav. Suppl. **90**: 146, 1938.
3. Beecher, Henry K.: Fatal Toxic Reactions Associated with Tribromethanol Anesthesia, J. A. M. A. **111**: 122, 1938.
4. Brown, A. L., and Archibald, E.: The Action of Cough Upon Material in the Tracheobronchial Tract, Am. Rev. Tuberc. **16**: 111, 1927.
5. Crafoord, Clarence: On the Technique of Pneumonectomy in Man, Stockholm, 1938, p. 61. Tryckeri Aktiebolaget Thule.
6. Lennon, B. B., and Rovenstine, E. A.: Fatality Following Rupture of Inflatable Cuff on Endotracheal Airway, Anesth. and Analg. **18**: 217, 1939.
7. Rovenstine, E. A.: Cyclopropane in Thoracic Surgery, Anesth. & Analg. **14**: 270, 1935.
8. Smith, C. A.: The Effect of Obstetrical Anesthesia Upon the Oxygenation of Maternal and Fetal Blood, With Particular Reference to Cyclopropane, Surg., Gynec. & Obst. **69**: 584, 1939.

*Dr. E. D. Churchill, surgeon. Data to April 1, 1940, covering a ten-year period.

DISCUSSION

DR. HARRY R. DECKER, Pittsburgh, Pa.—We have listened with profit to an admirable and timely paper by Dr. Beecher, from the clinic of one of the most successful surgeons of our Association. When these gentlemen speak, they do so with authority, based on careful observations and study of substantial material. We are almost persuaded by Dr. Beecher's presentation to his viewpoint of anesthetic agents and technique.

TABLE I

ANESTHETICS

	AVERTIN	SODIUM PENTOTHAL	ETHER	NITROUS OXIDE	CYCLO-PROPANE	SPINAL	LOCAL
Toxicity { Liver, Kidney, Heart }	4	2	2	1	2	1	1
Depression of respiratory center	3	3	1	2	1	3	1
Anoxia	3	2	2	4	1	2	1
Dosage control	4	2	2	1	1	2	1
Recovery cough reflex	4	2	3	1	1	2	1
No elevation of blood pressure	2	1	1	3	1	1	1
Quiet respiration	1	1	3	3	1	1	2
Irritation	1	1	4	2	1	1	1
Psychic shock	1	1	1	1	1	3	4
Inflammability. Explosibility	1	1	4	2	4	1	1
Total points	24	16	23	20	14	17	14
Safety points	20	12	14	11	10	11	6

1, Excellent; 2, good; 3, fair; 4, poor.

In an effort to reduce to graphic form our own experience with anesthesia, in the thoracic field, Table I was constructed in which are recorded the merits and demerits of the various agents we have used, graded on a scale of 1 to 4, as being excellent, good, fair, and poor. In summation, it is seen that of the barbiturates, sodium pentothal, given intravenously, is much to be preferred to avertin; of the gases, cyclopropane to nitrous oxide or ether vapor. Local anesthesia is to be preferred to all other forms if safety is the only standpoint. Our experience with avertin is quite like that of Dr. Beecher. We consider it a toxic and dangerous drug. We do not like ether because of a definite irritation to the bronchial mucosa and its prolonged recovery time. Nitrous oxide alone does not give proper relaxation and depth of anesthesia, and when supplemented for this purpose by ether and oxygen, is still responsible for a high degree of anoxia. Whereas spinal anesthesia, if carried out by the technique of Sise, is our preference for general surgery below the diaphragm, in thoracic work we have found an element of risk added on account of the paralysis of the intercostal muscles, which, when added to the open pleura, interferes seriously with patient's ability to keep the bronchial tree free of secretion by coughing. We had one patient with advanced bronchiectasis who literally drowned in his own pus before the operation began.

In our opinion, local anesthesia has an indispensable place in thoracic operations, namely, for routine anesthetization of the chest wall to reduce or prevent shock. To its limitations, as Dr. Beecher has stated them, there might be added the factor of psychic strain and shock. A patient may stand one operation well with local anesthesia, but when called upon to undergo a second or third, is considerably upset mentally and nervously.

We have a definite partiality for cyclopropane for its well-known fine qualities to supplement local novocain anesthesia. Cyclopropane has particular merit because it can be given with 80 per cent oxygen, which reduces anoxia to a minimum. Induction is quiet, respiration quiet, and recovery speedy, in from three to ten minutes. Blood pressure is not raised appreciably. There is negligible irritation. Liquids can be taken soon by mouth without nausea or vomiting. There is no postoperative depression. It has proved safe for the patient in low concentration and free from toxic organic or cardiac effects as far as we have observed. We are conscious, however, of its high explosibility against which special precautions must be always taken. It may be that experimental studies with helium mixtures now in progress at the United States Bureau of Mines, in Pittsburgh, will develop an explosive-proof combination.

In dealing with pulmonary tuberculosis, we have always felt that inhalation anesthesia is detrimental, particularly if ether is used. So we have studiously avoided inhalation anesthesia for thoracoplasties and allied operations, and have chosen to supplement local anesthesia with barbiturates. At first, we tried sodium amytal intravenously, and then avertin, but have discarded both as being too toxic and uncontrollable. In the last year, we have been using sodium pentothal intravenously. Gauged by the estimates of our table, it rates high from the standpoint of safety, efficiency, and low anoxia, notwithstanding some adverse reports of its cumulative toxic effects on heart muscle and liver in laboratory animals. In a very limited experience, approximately fifty thoracoplasties, for the most part in cases of pulmonary tuberculosis at the Tuberculosis League Hospital, we have been well satisfied. With sodium pentothal, induction is speedy and smooth, a matter of seconds. This makes an especial appeal to the patient. By careful attention to the dosage given, cough reflex is obtained within a few minutes of the close of the operation. Many of our patients are able to cough, if urged, on return from the operating room. The average time of return of consciousness from the close of operation has been thirty minutes. This period would be reduced considerably if caffeine or picrotoxin were administered routinely as the anesthetic is discontinued. Dosage has been from 8 to 17 grains for operations lasting thirty to sixty minutes; the average has been 11½ grains. There is rarely nausea or vomiting, so that fluids given early by mouth will be retained. Our experience indicates that the drug has a good safety threshold. By watching the pulse and respiration, dosage is controlled satisfactorily. In none of our thoracoplasties has there been an accident or fatality or evidence of spread of infection attributable to the anesthetic. Whether sodium pentothal will continue to merit our confidence, the future will tell.

Time does not permit me to do more than second Dr. Beecher's observations on the wisdom of using endotracheal and positive pressure technique when the operation opens the pleura or is in danger of doing so.

In conclusion, it would seem impossible to expect universal agreement, either by surgeons or anesthetists, as to proper anesthetic or preferable method of administration for thoracic operations. However, it seems to me that enough factual anesthesia experience has been built up to bring about nearer unity of opinion where divergence and controversy now exist, and even to warrant a certain amount of standardization of agents and technique. It might be that spinal and avertin anesthesias should be banned. Would it not be a step forward for members of this Association to present anesthetic statistics to a responsible committee for correlation and a periodic report? It seems to me that such data would be a very helpful guide to our anesthesia judgment and practice, would minimize the too often tragic trial-and-error methods, and would insure a higher percentage of success in our surgery.

DR. GUSTAF E. LINDSKOG, New Haven, Conn.—I want to thank Dr. Beecher for the opportunity of reviewing his paper beforehand and of hearing him give his very well-considered presentation of a complicated subject.

I am sure we all agree that the anesthetist must receive a very heavy share of credit for success in the completion of visceral thoracic surgery. I do not think it necessarily follows that gross operative mortality is a satisfactory criterion of perfection in anesthesia. Certainly a very low mortality is an indication of successful surgery and also of successful anesthesia, but in the opposite direction, high mortality does not allow this easy interpretation. There are too many factors involved. There is the matter of operability, the matter of preoperative preparation, the matter of adaptability of operation, surgical technique, and the postoperative care.

Moreover, there are accidents and morbidity factors which do not appear in mortality statistics. The length of convalescence and the freedom of convalescence from various complications may be directly or indirectly related to anesthesia. I think, as Dr. Decker does, that this type of correlation and analysis of case material is probably of more importance than simple mortality statistics.

In our own relatively small series of lobectomies which were done for bronchiectasis, for cystic disease, and for chronic lung abscess, all were carried out with basal avertin, intratracheal intubation, and cyclopropane-oxygen mixtures. In this group there was a 4 per cent mortality, but we are still willing to admit that avertin has very definite defects, as well as advantages.

First, and probably foremost, is the prolongation of the recovery period with the inhibition of cough, sometimes during a very considerable period. This can be alleviated only in part by postoperative medication. Second is the initial cardiovascular depression, which is sometimes marked and probably contributes to the necessity for supportive methods, including transfusions, in the longer operations. However, with this combination, we have a smooth induction, and because of the respiratory ease which we have observed, a sufficiently quiet operative field; we have not been impelled to employ "controlled" ventilation, which we consider a potentially dangerous procedure. Only rarely have we used temporary phrenic block through the chest. It seems to me it is more desirable for the surgeon to learn to develop good techniques and habits of dissection in a moving field than it is for the anesthetist to completely immobilize a normally rhythmic field of movement.

Our statistical studies agree with Dr. Beecher's statement that the type of anesthesia has no direct relation to postoperative complications.

We use nitrous oxide only rarely, in those cases where the use of cautery seems indispensable and a local novocain anesthesia is inadequate.

I should like to hear from Dr. Beecher more definitely about the relative explosibility of ether-oxygen and cyclopropane-oxygen mixtures. Is it not a fact that increasing the oxygen concentration raises the explosibility of the former, as with most combustible gases, so that early impressions of ether safety may have to be somewhat revised?

DR. LEO ELOESSER, San Francisco, Calif.—Attempts at thoracic surgery in various outlying places, both domestic and foreign, have forced upon me the opinion that the end of anesthesia in thoracic surgery has not yet come. At home and under proper conditions, the anesthetist is, if not *the* most important member of the team, one of the most important, as Dr. Beecher's excellent results and his splendid paper show.

Any attempt to dispense with intratracheal insufflation I think will reduce us to maneuvers that are much simpler than those we have been discussing at this meeting. However, Dr. Graham's admonition forces us to the conclusion that we have to cast about for methods of anesthesia other than those that require the services

of an expert insufflation anesthetist and an expert team, because it may be that we will be forced to operate under conditions other than those that we have been accustomed to meet in our clinics.

To make things very short: Avertin is a good anesthetic as far as the operation goes, but it has always frightened me afterward because of the difficulty of arousing the patient.

Pentothal, if one is not called upon absolutely to maintain rhythmic respiration in the open chest, if it is administered by the drop method in small concentrations, has proved valuable in many cases if supplemented by other anesthetics, such as morphine, barbiturates, etc. It should be given quite slowly, drop by drop, as in the Mayo Clinic.

To me, it seems paramount in open chest operations under insufficient anesthesia to abolish the cough reflex. You can carry along, as the Japanese are in the habit of doing, with a wide-open pneumothorax and operate in it with comparative ease and safety as long as the man does not cough; but with the first cough he disestablishes his equilibrium, the mediastinum begins to flap, and you will never regain the equilibrium you had in the beginning. Also it seems to me that with a good anesthetist you can carry your anesthesia through safely and quietly if you will abolish the cough reflex at the outset.

By the use of liberal amounts of novocain in certain places, I think the surgeon is helped considerably. In the first place, if pleural adhesions are very thick, by injecting weak solutions of novocain in one-quarter or even one-eighth per cent dilution, you can accomplish dissection very easily, making the novocain do the dissection for you. In addition, if you inject novocain in about 0.5 per cent or 0.75 per cent dilution into the hilum of the lung, the cough reflex will be abolished almost in the beginning and much less anesthesia will be required to proceed.

I think those points may be of some value to us in case we are called upon to operate on the open chest under conditions other than those we have met in our clinics.

DR. FRASER B. GURD, Montreal.—I think that we have already said enough about our experiences with spinal anesthesia to make it wise, if not imperative, to say a word this afternoon.

In the first place, we again should make certain memoranda. Our clinic at the Grace Dart Home Hospital in Montreal is not a large one. As its name suggests, for nearly fifty years it was a home to which patients suffering from pulmonary tuberculosis were admitted to die. About five years ago, the hospital that some of you saw in the moving picture on Thursday afternoon was built, and an operating room was built into the institution. The medical staff asked me if I could provide a staff that would be willing to operate on at least a certain proportion of those patients, and I said I was quite willing to do so.

They were, in our opinion, the type of case which many clinics on the continent would hesitate or positively refuse to have anything to do with. Actually, of the approximately 100 cases that we have treated, of those that we have operated upon by means of thoracoplasty, only thirteen have been cases of unilateral disease; forty have been cases of bilateral disease with relative inactivity, and forty-six have been cases of bilateral active disease.

During the first two years that we worked with these cases, we met with a 31 per cent mortality rate in our first group of twenty-seven cases. The immediate cause of death in nearly all the cases was postoperative spread, either in the sense of an acute pneumonic phthisis or reactivation of the bilateral disease.

We came to the conclusion that that situation was intolerable, and in conference with Dr. Bourne, who had been giving our anesthetics, and we used the differ-

ent combinations, I asked him if he did not think we might do better with spinal anesthesia. Since that time, almost exactly three years ago, we have been using spinal anesthesia, during which time we have operated upon seventy-odd cases of the same type of late, for the most part bilateral, pulmonary tuberculosis, and we have reduced the case mortality rate to 13 per cent and the operation mortality rate to 4 per cent.

In addition to the cases at Grace Dart, in our general surgical practice at the Montreal General we employ spinal anesthesia for all thoracic surgery, with special reference to pulmonary abscess, in which the necessity for stabilization of the mediastinum does not appear to be an important matter.

We are still using, and shall continue to use, spinal anesthesia for the type of case that it is our fortune or misfortune to treat in the Grace Dart Home Hospital.

DR. WILLIAM E. ADAMS, Chicago, Ill.—I have enjoyed very much hearing Dr. Beecher's paper. I think the Association should feel very fortunate in having a man with his knowledge of the physiology of the cardiorespiratory system as applied especially to anesthesia discuss this subject before it. I think he sounded the keynote when he stated that simplicity has its place in this type of work. I know of no other field in surgery where cooperation between the anesthetist and the surgeon at all times during the operative procedure is more needed.

There are just a few factors that I wish to mention, and the first of these is anoxia. So much emphasis has been placed upon either the type of anesthesia, or the method in which it is given and the materials, that during the course of the operative procedure disturbances of the oxygen-transporting mechanism by alteration of the intrathoracic pressure relationship are not always recognized as such. All too frequently the chief thought concerns adequate ventilation of the lungs under plenty of pressure, with little appreciation of the hazards that accompany its use. The entire mechanism of oxygen transportation must be kept in mind, ventilation of the lungs being only the first step of this process.

During an operative procedure within the chest, pressure on the great veins or on the auricles by gauze packs or retractors is likely to cause a considerable reduction in cardiac output, with resultant signs of anoxia; the surgeon may say, "A little more pressure," or "More oxygen. The patient is cyanotic," when, as a matter of fact, the surgeon is causing the trouble himself.

This brings us to the question of intrabronchial pressure. Where there are no adhesions within the pleural cavity, as Dr. Beecher said, a small amount of pressure will be needed in most open chest operations. Now, it is natural that one should gain impressions according to the type of operations that are being performed. That not a great amount of intrabronchial pressure may sometimes lead to a considerable amount of trouble, either during or after operation, has been seen in an ever-increasing number of reports of serious accidents, some terminating fatally.

I would like to show you a few slides to demonstrate these points. (Slide) On the left is an x-ray of a young girl who had an operation on the stellate ganglion for circulatory disturbance of the upper extremity. The operation was on the right side, and during the procedure the neurosurgeon made a small opening in the parietal pleura. He asked the anesthetist to increase the pressure. It so happens that this surgeon always wanted intratracheal pressure anesthesia. The anesthetist gave him about 10 or 12 mm. mercury pressure, distended the lung, and the wound was closed.

During the same day, late in the evening, this patient became dyspneic, and it was found that she had developed a mediastinal and subcutaneous emphysema, with a pneumothorax on the left side and collapse of the left lung. About 1500 c.c. of

air were removed from the left chest, and normal respiration was immediately restored. The following day another liter of air was removed from the left side, and she made an uneventful recovery. That this is not an infrequent complication of intratracheal anesthesia is indicated by occasional reports in the literature and several personal communications. On the right side of the slide is an x-ray of the chest following complete recovery.

(Slide) Dr. Marcotte and I studied the effect of positive intrabronchial pressure on the circulation in dogs; here we see various amounts of intrabronchial pressure used, and above, the effect on the general circulation. With as little intrabronchial pressure as 8 or 9 mm. of mercury, a fall in systolic blood pressure of between 10 and 20 mm. of mercury resulted, and as the pressure in the intrabronchial system increased, the fall in the circulatory system did likewise.

(Slide) As stated before, the fact that one may be dealing with different types of operations may determine to a great extent the type of anesthesia preferred. We have used the intratracheal catheter method of administration occasionally, but for the most part, the simple face mask with an ethylene-oxygen mixture has been employed. Adequate inflation of the lungs is easily obtained by the latter method; if intermittent suction is needed to remove bronchial secretions, the intratracheal catheter method may be indicated.

(Slide) This will give you some idea of the cross section of the types of operations, all of which were performed with a wide-open chest. The series includes large tumors of the chest wall, mediastinal tumors, etc. The sympathectomies performed were intrapleural, in which the entire thoracic sympathetic chain was removed on both sides.

Regarding the mortality following the operations, in some of these death was due to the condition itself. Lobectomy, bilobectomy, and trilobectomy (bilateral operation) for bronchiectasis (about twenty-five in all) were accompanied by no immediate or postoperative mortality.

(Slide) This is a tracing of the anesthetic record during the removal of a mediastinal tumor, using simple face mask anesthesia. On the left, the blood pressure and respiratory tracings are recorded, and, as you see, they remained fairly constant throughout the operation.

Thus, when considering the anesthetization of a patient for an endothoracic operation, one must consider the entire mechanism for oxygen transportation and not ventilation alone. Although we have used simple mask anesthesia for most of these operations and have felt perfectly satisfied with it, the chief thought I want to leave with you is that too much intrabronchial pressure may give rise to serious consequences and is more likely to occur with intratracheal catheter administration, but can also be produced by simple face mask technique. Thank you.

DR. BEECHER.—I want to thank those who have been good enough to discuss my paper.

In regard to Dr. Lindskog's remarks concerning the efficacy of avertin, I must confess I am still unable to understand what the advantages of avertin are, beyond the peace of mind the patient gets ahead of time and perhaps during induction. We have had three patients who could not speak English come into the general emergency ward, bearing little slips of paper which read, "Avertin." They know about it and like it. The advertising company has done a good job. But we believe the patient who is to undergo thoracic surgery pays too high a price for the pleasant avertin induction.

Dr. Lindskog asked about the relative explosibility of ether and cyclopropane. That is a complicated thing. We can rule out all explosion due to cigarettes and fires and cauteries and that kind of thing. So far, none of us knows enough to

guard against all possibilities of electrostatic explosions. That is the great hazard, I think, which worries us all so much.

Some objective evidence is obtainable in regard to the two agents on that basis. I have checked up with the American Society of Anesthetists; a committee of that organization has been looking for these explosions over a long period of time. The record goes back as far as the memory of some of the oldest living anesthetists. During that time (until a few months ago), according to the letter I received they had uncovered records of two persons, one a patient and one a bystander, who were killed by static electricity setting off an ether mixture. On the other hand, they reported to me that they had in their files records of eight patients who had been killed by static setting off cyclopropane. Since that time there have been two more widely publicized cyclopropane cases. That shows two* cases of ether against ten cyclopropane "static" deaths. Ether certainly has been used with closed anesthesia and high oxygen as long as cyclopropane. We do not know what the numbers are, I grant you, but certainly both have been used very widely. On the one hand, we have a mortality five times as high as on the other. Is that significant? I do not know; I think it may be.

In regard to Dr. Eloesser's statement that the end is not here, I heartily agree with him, and only say that this report is offered as a progress note. I am sure we shall all change a lot of things before the end will be in sight.

Dr. Graham pointed out to me yesterday that during the time of the last war many surgeons did not appreciate clearly the dangers of pneumothorax in handling chest wounds. Certainly we must do a better job of it this time. I am convinced that the simplification of these procedures of anesthesia for thoracic surgery as described here is not only important in civil life, but extremely important in the military situation which confronts us all.

In regard to Dr. Gurd's statement, I am very glad that he presented an opposing point of view. I appreciate it, even though I cannot agree with him. I only hope that I can personally discuss with him the bad risk thoracoplasties we have been doing under ether for a period of some five years.

*Since this discussion, I have learned of another ether death due to static.

THE OPERATIVE TREATMENT OF CARDIOSPASM

HOWARD K. GRAY, M.D., AND IRA C. SKINNER, M.D.* (BY INVITATION)
ROCHESTER, MINN.

CARDIOSPASM is the term most frequently used to imply spasm of the musculature of the cardia or epicardia sufficient to prevent completely or partially the passage of food from the esophagus into the stomach. The controversial nature of the etiology of the disease is indicated by the number of synonyms used by different authors to describe it. Achalasia of the cardia, simple ectasia of the esophagus, preventriculosis, hiatal esophagismus, phrenospasm, paralytic dilatation of the esophagus, mega-esophagus, spindle-shaped dilatation of the esophagus, and idiopathic dilatation of the esophagus are some of the terms found in the literature.

Willis, as early as 1674, described a typical case of cardiospasm and recommended treatment which foreshadowed that used today. He reported the following interesting observations in his "Pharmaceutica rationalis."

> No less will a very rare case of a certain man of *Oxford* show, an almost perpetual vomiting to be stirred up by the shutting up of the left orifice. A strong man, and otherwise healthful enough, labouring for a long time with often vomiting, he was wont, very often, though not always presently to cast up whatsoever he had eaten. At length the disease, having overcome all remedies, he was brought into that condition, that growing hungry he would eat until the *oesophagus* was filled up to the throat, in the meantime nothing sliding down into the ventricles, he cast up raw (or crude) whatsoever he had taken in: when that no medicines could help and he languished away for hunger, and every day was in danger of death, I prepared an instrument for him like a rod, of a whale bone, with a little round button of sponge fixed to the top of it; the sick man having taken down meat and drink into his throat, presenting putting this down in the *oesophagus*, he did thrust down into the ventricle, its orifice having opened, the food which otherwise would have come back again; and by this means he hath daily taken his sustenance for fifteen years and doth yet used the same machine, and is yet alive, and well, who would otherwise perish for want of food. Without doubt in this case the mouth of the stomach being always closed, either by a tumour or Palsie, nothing could be admitted into the ventricle unless it were violently opened.

The next case was probably that of Hoffmann in 1733, the title of his article being "De spasmis gulae inferioris et de nausea." Purton, to whom most authors ascribe the first published case, presented his in

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From the Division of Surgery, Mayo Clinic.
*Fellow in Surgery, The Mayo Foundation.

1821. Hannay in 1833, also described a case. In 1876, Zenker and von Ziemssen were able to collect seventeen cases from the literature. In 1900, Mikulicz estimated that approximately 100 cases had been reported in the literature. In 1908, Plummer[23] reported forty cases from the Mayo Clinic, and in 1912,[25] 156 cases. Since then, this group has grown so that it now includes more than 1,200 cases. The extensive literature on cardiospasm was reviewed in 1933 by Sturtevant.

ETIOLOGY OF CARDIOSPASM

The etiology of cardiospasm is still in dispute. The disease has been attributed to primary atony of the musculature of the esophagus, to spasm of the cardia, to failure of proper relaxation of the cardiac sphincter, to spasm of the diaphragm, to pressure on the esophagus by the left lobe of the liver, to kinking of the esophagus, to pressure from the lower lobes of the lungs, to fibrosis of the periesophageal connective tissue, and so forth. Recent pathologic and experimental work have made the theory of autonomic imbalance the most acceptable one. Knight, and Knight and Adamson produced a condition in cats simulating cardiospasm by bilateral sectioning of the vagus nerves. Simultaneous removal of the sympathetic fibers prevented the onset of the obstruction. They also showed that after obstruction had developed, it could be relieved by section of the sympathetic nerves supplying the sphincter. Further anatomic work indicated that in the human being the sympathetic supply to the cardia of the stomach passed to the sphincter along the course of the celiac axis and left gastric artery and, therefore, was readily accessible to surgical approach.

Rake first described degeneration of the cells of Auerbach's plexus in cases of cardiospasm. Other investigators,[2, 18] subsequently noted these pathologic changes in the cells. Hill considered the cells of Auerbach's plexus to be vagal relays. Therefore, it seems feasible that in some cases of cardiospasm at least the vagal supply to the cardia is interrupted and unrestricted activity of the sympathetic nerve supply results.

PATHOLOGIC CHANGES PRODUCED BY CARDIOSPASM

A study of the gross pathologic changes of the esophagus produced by cardiospasm demonstrates why all cases are not amenable to simple dilatation. The characteristic gross pathologic changes seen are dilatation of the esophagus, hypertrophy of its wall, and actual lengthening of the organ, with resultant tortuosity. The dilatation usually assumes one of three typical shapes, as shown by Lambert: fusiform, flask-shaped, or sigmoid-shaped (Fig. 1). In the fusiform variety the lumen of the esophagus increases to a point midway between the cricoid cartilage and cardia, and then gradually decreases in size (*a*); in the flask-shaped type the dilatation is immediately above the cardia (*b*). In both of these

varieties the cardia is the most dependent portion of the dilated esophagus and both are readily relieved by dilatation from above. The sigmoid-shaped esophagus (*c*) represents an advanced pathologic change and fortunately is the rarest type. Lambert described its formation about as

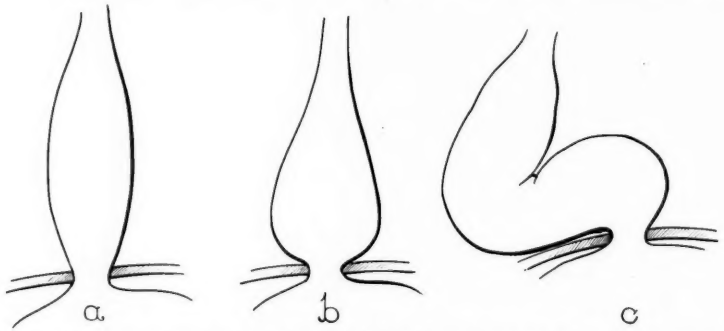

Fig. 1.—Various types of esophagi associated with cardiospasm, after the manner of Lambert: *a*, fusiform; *b*, flask-shaped; and *c*, sigmoid-shaped.

follows: The upper end of the esophagus opposite the cricoid cartilage and the lower end at the cardia being more or less fixed points, the increased length of the esophagus accompanying advanced dilatation is accommodated by a curved course. The dependent point of the dilated esophagus rests on the diaphragm to the right and posterior to the cardia; it then ascends to the left and enters the abdomen through the diaphragm at a higher level. Thus a reservoir is developed below the level of the cardia. In the sigmoid form, dilation from above is not only difficult to accomplish but will fail to remedy the mechanical derangement unless the esophagus recovers sufficient tone to empty its reservoir. It is in such cases that operative treatment will be found necessary.

SYMPTOMS OF CARDIOSPASM

According to Plummer,[23] the symptoms of cardiospasm are characteristic of the stage of the disease. He found three stages, more or less clearly defined, in its clinical course: (1) cardiospasm without regurgitation of food; (2) cardiospasm with immediate regurgitation of food; and (3) cardiospasm with a dilated esophagus and retention of food in the dilated part, with this undigested food regurgitated at varying intervals. The characteristic symptoms are dysphagia, regurgitation, and epigastric pain. Respiratory disease may result from the inspiration of the contents of the esophagus. The dysphagia is usually of long duration and is as marked for liquid as for solid foods. Regurgitation may occur immediately after meals or may be delayed for hours; this depends on the amount of food taken, the degree of dilatation, and the tone of the esophagus. Nocturnal regurgitation is a particularly disturbing feature.

Epigastric and substernal pain may precede the appearance of dysphagia by many months and must be differentiated from that caused by disease of the biliary tract, angina pectoris, peptic ulcer, and paraesophageal hernia.

According to Plummer and Vinson, the diagnosis of cardiospasm may be made with relative certainty if the patient has had dysphagia for five or six years without previous trauma to the esophagus and if roentgenograms reveal a smooth cigar-tip type of obstruction at the cardia, with or without dilatation of the esophagus. The passage of a No. 45 French olive into the stomach guided by a previously swallowed silk thread, with only slight resistance at the cardia, confirms the diagnosis. Esophagoscopy is usually not necessary, but occasionally is helpful in doubtful cases.

TREATMENT OF CARDIOSPASM

Dilatation of the cardia from above by some type of mechanical dilator is an extremely satisfactory method of treatment in most cases of cardiospasm. The early efforts of such men as Russel, Plummer,[23] and Sippy in this field made this type of treatment possible. In more than 1,200 cases of cardiospasm encountered at the Mayo Clinic, operative measures have been resorted to in only seven cases. Moersch, in 1933, reported 810 cases observed at the clinic. Of these, 805 were treated conservatively. Seventy-one per cent of the 670 patients traced were completely relieved. A large proportion of the remainder were benefited; only 32 received no help. Nine died as a result of splitting of the esophagus. All but one of these deaths occurred prior to 1925; since then there has been a change of technique. Such statistical studies prove the extreme efficaciousness of the nonoperative treatment. However, in a small group of cases dilatation will not prove beneficial because of the deranged mechanics of the dilated and tortuous esophagus. It is this group of cases that we wish to present here.

Surgical Treatment.—Many very interesting operations have been devised by various surgeons for the relief of cardiospasm. Perhaps the first was that of Mikulicz who, in 1900, performed gastrotomy on a girl to dilate the cardia. His procedure consisted of the introduction of a long, curved forceps with rubber-covered blades into the cardia through an opening in the wall of the stomach, after the cardia had been dilated manually sufficiently to receive the blades of the forceps. The blades of the forceps then were opened gradually until the maximal distance between them was 6 or 7 cm. This procedure is similar to the present method of dilating the cervix. In 1904, he reported four cases in which this type of treatment was employed. In two, operation had been performed more than one and one-half years prior to his report, and in two, approximately nine months before. In all of these, cure resulted. Since 1904 this operation has been performed many times by various surgeons,

usually with good results. Today manual dilatation of the cardia is preferred to the use of the blades of the forceps (Fig. 2). Walton used this method of treatment in the largest group of cases. He reported 22 cases in which digital dilatation through the opened stomach was employed. In 14 of these the previous passage of bougies had failed to give relief. Three deaths occurred. Of the remaining 19 patients, 13 remained in perfect health. Lindström and Kümmel have dilated the cardia without opening the stomach. The wall of the stomach was simply invaginated into the cardia by the dilating fingers.

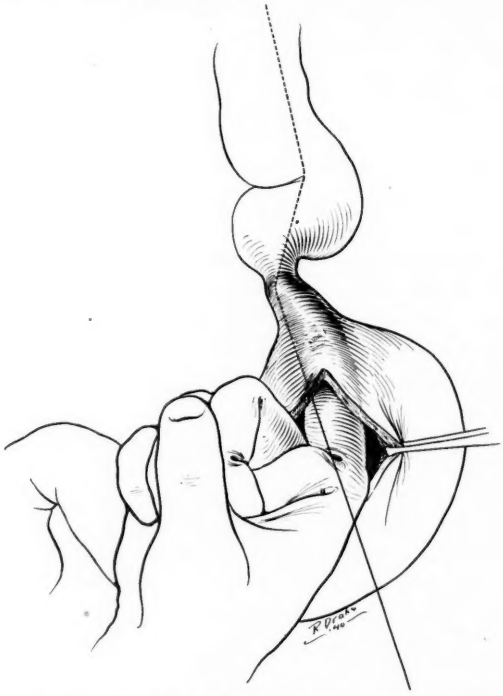

Fig. 2.—Retrograde dilatation of the cardia for cardiospasm.

From a study of the literature, esophagogastrostomy seems to be a very satisfactory operation in certain cases of cardiospasm which are resistant to treatment by dilatation. We found this true in one of our cases. The transpleural and transperitoneal approaches to the esophagus have been used. Heyrovsky, in 1910, performed the first esophagogastrostomy by the abdominal route. Bull, in 1925, collected 16 cases from the literature in which this method was employed, with only one death. In two cases the results were not good, but in the remainder they were entirely satisfactory. Häggström, in 1930, collected 29 cases in which esophagogastrostomy was performed by the transperitoneal route. He stated that

all of these operations were successful, whereas when the transpleural route is used, two of every five patients die.

The technique of abdominal esophagogastrostomy has been described by various surgeons.[1, 31, 36] For the debilitated patient preliminary gastrostomy or jejunostomy has been recommended for feeding purposes prior to the more extensive operation. At other times it is feasible to perform gastrostomy at the time of the major procedure, thus avoiding the increased difficulties of separating adhesions and the necessity of performing two operations. Satisfactory exposure can be obtained through a left rectus incision which begins in the left costoensiform angle and extends a little beyond the umbilicus. If necessary, the left lobe of the liver can be mobilized easily by severing the left lateral ligament and then conveniently packed away to the right beneath the abdominal wall. The spleen is likewise retracted downward and to the left by a large, specially designed retractor. It is probable that preliminary interruption of the left phrenic nerve, as in cases of diaphragmatic hernia, would facilitate the operation, although we can find no report in the literature of this having been done.

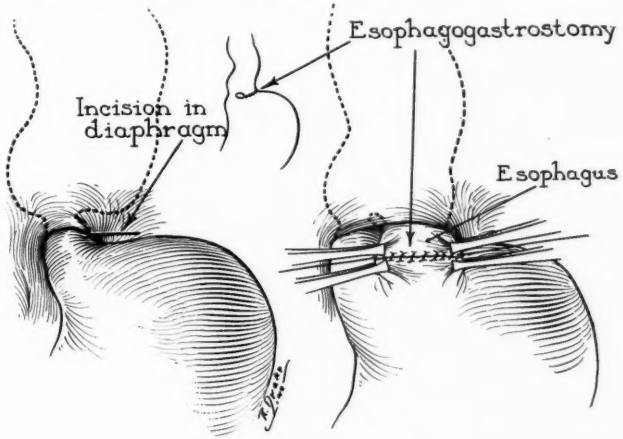

Fig. 3.—Esophagogastrostomy.

When satisfactory exposure has been obtained, the stomach is drawn well down by means of a right angle rubber-covered clamp, and the abdominal portion of the esophagus which is covered by peritoneum is brought into view. The peritoneum is divided at the point where it is reflected from the diaphragm onto the esophagus. Vessels running along the anterior margin of the hiatus should be avoided. A finger is introduced into the mediastinum and as much of the thoracic portion of the esophagus as possible is mobilized. To accomplish this, the esophageal hiatus may be split a short distance to the left (Fig. 3). The finger is then hooked around the freed esophagus and it is drawn down into the abdomen as far as possible. There is rarely difficulty in obtaining sufficient length of esophagus, particularly when the esophagus has assumed the sigmoid shape, as the organ usually has become lengthened as a result of the disease. The esophageal hiatus is attached high along the esophagus by interrupted silk sutures to prevent retraction of the anastomosis into the thorax and to lessen the likelihood of soiling the mediastinum at the time of the anastomosis. The anterior portion of the fundus of the stomach

is anastomosed to the redundant esophagus as in gastroenterostomy (Fig. 3). The incision in the esophagus may be extended in an arc through the cardia and the union accomplished in the manner of a Finney[1, 40] pyloroplasty (Fig. 4). The left lobe of the liver falls into place over the suture line and adds to its security. A drain may be placed down to the anastomosis if there is fear of contamination.

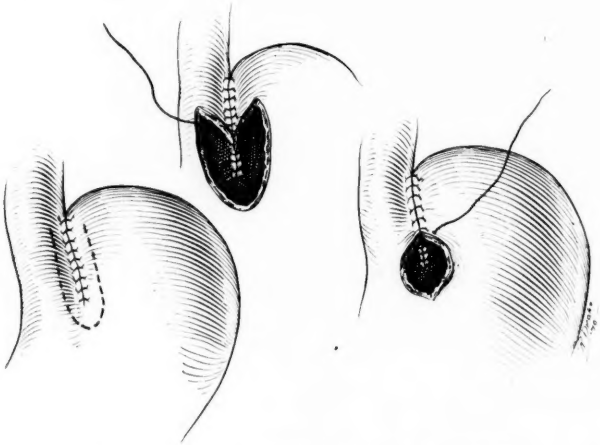

Fig. 4.—Cardioplasty after the manner of Womack.

Lambert has accomplished esophagogastrostomy by means of a clamp. After gastrotomy, he passed one blade of the clamp through the cardia into the dilated portion of the esophagus lying within the abdomen and the other blade of the clamp remained within the stomach. The clamp was then closed sufficiently to hold firmly but not to cut through the structures included between the blades. The handle of the forceps was brought out through the opening in the wall of the stomach and the abdomen. On the fourth day the clamp was tightened to its full extent and the spur between the stomach and esophagus was crushed. The clamp was removed on the ninth postoperative day after an opening had been established between the esophagus and stomach.

The nerve supply of the cardia has been attacked by several operative procedures in an attempt to relieve cardiospasm. Clairmont proposed sectioning of both vagi at the lower end of the esophagus. Craig, Moersch, and Vinson reported a case of cardiospasm associated with intractable pain which was relieved temporarily by bilateral cervicothoracic sympathectomy. The recent work of Knight[14] has done much to clarify the nerve control of the cardia. Following his experimental work, Knight and Adamson[15] interrupted the greater portion of the sympathetic nerve supply to the cardia in four cases by removing a segment of the left gastric artery along with the mass of nerve tissue that accompanied it (Fig. 5). Recently Eliasan and Erb have reported two

cases and Meade has reported one case in which this type of treatment was used. In one of our cases (Case 5), sympathectomy together with manual dilatation of the cardia was employed.

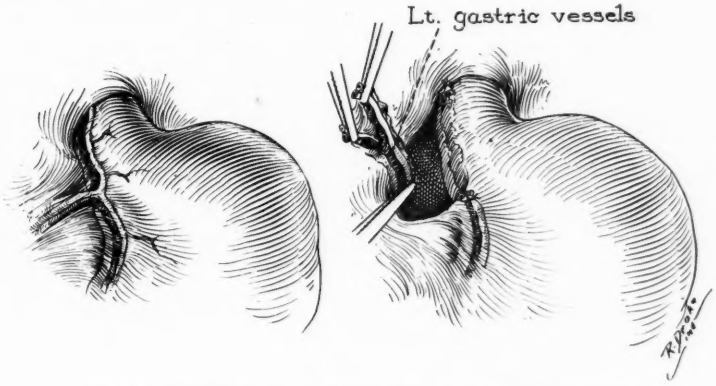

Fig. 5.—Denervation of the cardia after the method of Adamson.

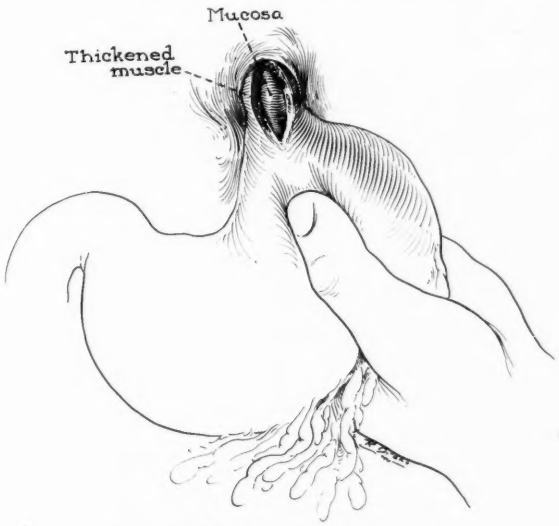

Fig. 6.—Cardioplasty after the method of Heller.

Heller performed the operation of extramucosal cardioplasty based in principle on Ramstedt's procedure for congenital hypertrophy of the pylorus. This operation consists in splitting the muscular coat of the cardia down to the mucosa (Fig. 6); anterior and posterior incisions were made. In 1921 Heller reported at the German Surgical Congress 21 such operations performed by various surgeons, which were success-

ful in 17 cases. The results were unsatisfactory in four cases, and there were no deaths. More recent reports have not been so favorable. Schaldemose modified this procedure, making a circular incision 1 cm. above the cardia through the muscular wall into the submucosa. Roepke removed connective tissue, nerves, and muscle down to the mucosa from the cardia upward until the dilated portion of the esophagus was reached. Cardioplasty consisting of a vertical incision through all the walls of the cardia repaired in a transverse manner has been used with success in a few cases.

Seneert and Simon proposed that the esophagus be freed from its cellular bed, drawn down as far as possible into the abdomen, and then resutured to the hiatal margins; the object was to straighten the tortuous esophagus. In addition they performed cardioplasty. Turner freed the esophagus, drew it down into the abdomen, and resutured it to the diaphragm in five of his cases, with lasting results in only one. Willy Meyer, in 1911, performed esophagoplication by the transpleural route, with relief of the condition in one case. .Freeman shortened the esophagus in one case by intussusception of the redundant length by manipulation through a cervical incision; cure resulted.

Plummer[26] reported the following interesting case in 1906.

CASE 1.—A married woman, aged 58 years, was first examined Aug. 6, 1905. She stated that she had been in perfect health until four and one-half years prior to her admission to the clinic. At this time she began to suffer from periodic episodes of dysphagia. For three years the trouble had been practically continuous. Spasms of severe pain would occur after she had eaten her meals. The greater part of the food was being regurgitated soon after it was taken. She had lost 50 pounds (22.7 kg.).

On examination the patient was found to be markedly emaciated. The remainder of the physical examination gave essentially negative results. A stomach tube could be passed into the stomach only after repeated attempts, but a large sound could be passed easily. A roentgenogram revealed a large cylindrical dilatation of the esophagus, 3 in. (7.6 cm.) in diameter, extending from the diaphragm to the upper border of the sternum. The diagnosis of cardiospasm associated with dilated esophagus was made.

On Aug. 23, 1905, Dr. W. J. Mayo operated upon the patient, performing gastrostomy and dilatation of the cardia after Mikuliez's method. This gave immediate relief. Four months later the dysphagia returned but was less annoying than before the operation.

Judd, Vinson, and Greenlee in 1929 published descriptions of two cases in which retrograde dilatation of the esophagus was performed. A previously swallowed thread was used as a guide to the cardia. Their cases were reported as follows:

CASE 2.—A man, aged 60 years, came to the Mayo Clinic on Jan. 19, 1925, complaining of dysphagia of thirty-eight years' duration. He dated the onset of trouble to drinking cold lemonade, which caused a tight substernal sensation and was re-

lieved by regurgitation of the lemonade. Since then cold liquids or solids always seemed to lodge under the lower end of the sternum. At times he was able to force food down by taking a large amount of fluid. The trouble had persisted to a greater or lesser degree since the onset. He coughed and became blue with attacks of marked dysphagia and frequently regurgitated large amounts of mucus. He regurgitated food at night.

The patient weighed 145 pounds (65.8 kg.), which represented a loss of 15 pounds (6.8 kg.) compared to his weight at the onset of the trouble. A roentgenogram of the esophagus revealed cardiospasm with tremendous dilatation of the esophagus.

Several attempts were made to dilate the cardia with a hydrostatic dilator, but the lower end of the esophagus was so angulated that it was impossible to introduce the instrument into the cardia, even with the guiding thread. It was then decided to dilate the cardia manually from an approach through the stomach.

On Aug. 10, 1927, exploration was carried out and gastrotomy performed. Following the thread, one finger and then two were introduced into the cardia, thus dilating the esophageal opening considerably. The patient was relieved of dysphagia, and fluoroscopic examination revealed slight lagging of the barium meal at the cardia. After nine months mild symptoms recurred. The patient returned for re-examination in June, 1928, and at that time it was perfectly easy to introduce the hydrostatic dilator from above and effectually stretch the cardia. Complete relief followed this dilatation.

CASE 3.—A woman, aged 52 years, was first seen at the Mayo Clinic on Jan. 4, 1928. Her presenting complaint was abdominal pain of 30 years' duration. The symptoms at the onset consisted of gas, a sensation of fullness, and rather sharp upper abdominal pain which came on shortly after the taking of food. Seven years later urgent spontaneous vomiting without nausea developed. Her weight dropped from 105 to 90 pounds (47.6 to 40.8 kg.).

Fifteen years previously a roentgenogram had disclosed a pouch to the right of the esophagus. The condition was diagnosed as stricture of the esophagus and was dilated with bougies, without relief. At this time the patient was having severe attacks of pain which awakened her at night; this pain was associated with vomiting and required morphine for relief. Seven years before examination she had had hematemesis on two occasions a few days apart. Severe upper abdominal pain occurred frequently at that time and persisted at irregular intervals until admission.

On examination the patient was found to be moderately emaciated; she weighed 107 pounds (48.5 kg.). There was slight epigastric tenderness. The hemoglobin was 67 per cent (Dare); results of urinalysis and Wassermann tests on the blood were negative. A roentgenogram of the esophagus revealed cardiospasm, with marked dilatation and tortuosity of the esophagus. An attempt was made on seven occasions to dilate the cardia from above with the ordinary hydrostatic dilator, but it was impossible to introduce the dilator into the cardia. In view of this, dilatation of the esophagus from below was advised.

Exploration was carried out April 4, 1928. The stomach and gall bladder were normal. An opening was made in the stomach, and the thread, which had been introduced through the cardia into the stomach from above, was followed up to the esophageal opening. One finger was easily introduced into the cardia, but it was necessary to introduce the whole hand into the stomach to get two fingers into the cardia and dilate it. The opening in the stomach was closed with chromic catgut and silk sutures. The patient convalesced uneventfully, leaving the hospital 16 days after operation. A roentgenogram taken about three weeks after operation

revealed diminished dilatation of the esophagus and a good-sized opening into the stomach. Three months after the operation the patient was eating all types of food, without difficulty.

Craig, Moersch, and Vinson published the following case history in 1934:

CASE 4.—A married woman, 34 years of age, came to the clinic for examination on Sept. 8, 1934, complaining of difficulty in swallowing. The first discomfort she had experienced had been a dull pain over the precordium which had been projected down the left arm as far as the elbow. This had begun three years previously and had been followed soon by obstruction to all types of food at the cardia; the pain had also been associated with regurgitation after meals, and also during sleep. Fruits and cold liquids had been particularly difficult to swallow.

In addition to this dull pain, which had persisted for the greater part of the day, spasmodic epigastric pain had developed, and although it was of rather short duration, it had been very severe and had been projected to the neck, ears, lower jaw, and occipital region. This pain had frequently awakened the patient at about 4:00 A.M., and for a period of four months during the year before admission she had used tablets of morphine and codeine for relief. Phenobarbital (luminal) and belladonna had been used, without benefit. The fear of becoming an addict to the use of morphine had caused the patient to discontinue all types of medication although it had been difficult for her to tolerate the pain. For a time the ingestion of very hot water had provided temporary relief, but this had lost its beneficial effect. The patient had lost 13 pounds (5.9 kg.).

General physical examination revealed nothing abnormal. Roentgenoscopic study of the esophagus showed a smooth obstruction at the cardia, with moderate dilatation of the esophagus above the point of obstruction. The cardia was first dilated with a No. 60 French sound, and then, during the succeeding two months, eleven dilatations were done with a hydrostatic dilator. Swallowing was slightly improved by these dilatations, but both types of pain were apparently made worse by instrumentation. Esophagoscopy revealed considerable inflammatory reaction in the lower portion of the esophagus, without ulceration. With improvement in swallowing, the patient gained 20 pounds (9.0 kg.), but, because of the persistence of considerable dysphagia and the intolerable character of the pain, it was decided to attempt to interrupt the sympathetic nerve supply of the cardia, first by injection and later by ganglionectomy.

In view of the fact that it was impossible to make an injection during the paroxysmal type of pain, a diagnostic injection of procaine was advised to determine whether or not th severe pain could be initiated or the residual pain could be relieved. On Nov. 2 , 1934, after anesthetizing the skin with procaine, needles were inserted paravertebrally over the eighth cervical nerve, and first, second, and third thoracic nerves on the left side. A suggestion of the severe type of pain could be elicited when the needle was inserted against the transverse process of the first thoracic vertebra. The needles were then introduced in the region of the sympathetic chain, and 5 c.c. of a 1% solution of procaine was injected. During injection of the first and second thoracic sympathetic ganglia, the severe, spasmodic type of pain was reproduced; in a few moments it was relieved, as was the aching residual type of pain. After the injection, Horner's syndrome developed, and increase in temperature of the skin of the entire left upper extremity, side of the face, and ear was noted. In addition to this, there was some anesthesia of the skin and weakness of the left hand. The weakness and anesthesia disappeared in about 10 minutes, although the change in temperature persisted.

Following the injection, the patient was completely relieved of pain for one and one-half hours, and during this time she drank cold water and ate the type of food which had sometimes initiated the pain; however, she was completely free from symptoms. This indicated that the first and second thoracic sympathetic ganglia on the left side were involved in the mechanism of the patient's pain. The following day, the Horner syndrome had disappeared; the hand, arm, and face had resumed a normal temperature, and the pain had returned. The temporary relief of pain evidenced by the diagnostic injection of procaine was indicative of a permanent relief of pain following removal of the sympathetic ganglia.

On Nov. 24, 1934, with the patient under intratracheal anesthesia, the posterior mediastinum was entered on both sides through a posteromedian incision in the skin which extended from the tip of the fifth cervical spine to the tip of the fourth thoracic spine and resection of a portion of the transverse process and the first rib; the cervicothoracic sympathetic trunk and ganglia were removed on both sides. A bilateral operation was done to denervate the esophagus and to equalize the rise in surface temperature of the skin of both arms and both sides of the face. From a cosmetic standpoint also, the bilateral operation is preferable since it produces bilateral Horner's syndrome.

The patient's postoperative course was uneventful, and the wound healed perfectly. Following operation she was completely relieved of dysphagia and of the residual type of pain, and she has had no further attacks of the acute paroxysmal type of pain. The patient was permitted to return to her home on December 14.

CASE 5.—A farmer, 48 years of age, was first seen at the clinic on Oct. 18, 1934. When he was 13 years old, his mother had given him undiluted liniment to take for a throat ailment. Since that time he had experienced difficulty in swallowing. Food, especially solid food, seemed to stick in the lower end of his esophagus. However, he was always able to force the meal through by drinking large quantities of water. One month prior to his admission to the clinic the dysphagia increased and he began to regurgitate most of the nourishment taken. Worry over his condition had produced a rather marked mental depression. He had consulted his home physician. Fluoroscopic studies revealed the esophagus to be markedly dilated, and a presumptive diagnosis of cardiospasm was made. For the five days before coming to the clinic the patient had been unable to swallow solid or liquid foods. He was unable to walk because of weakness from lack of nourishment.

On examination the patient was found to be emaciated and dehydrated. His systolic blood pressure measured 106 mm. Hg and his diastolic pressure, 68 mm. Hg. His pulse was 60 beats per minute, and his temperature normal. Laboratory examinations revealed the hemoglobin of 16.1 Gm. per 100 c.c. of blood and 4,180,000 erythrocytes per c.mm. of blood. Leucocytes numbered 6,700, 85% of which were neutrophiles, 7 per cent monocytes, and 8 per cent lymphocytes. The concentration of urea was 58 mg. per 100 c.c. of blood, and of the chlorides, 644 mg. per 100 c.c. of plasma. A flocculation test for syphilis was negative. Roentgenologic examination of the chest revealed nothing abnormal. Fluoroscopic study of the esophagus after a barium meal disclosed complete obstruction at the cardia, with marked tortuosity and dilatation of the esophagus. The roentgenologic diagnosis was cardiospasm with complete obstruction.

On Oct. 19, 1934, esophagoscopy was carried out under local anesthesia. The esophagus was found to be markedly dilated and filled with a very foul fluid. This was aspirated thoroughly; a stomach tube was passed into the stomach and 1,000 c.c. of fluid were given. A thread with a shot attached was left in the lower end of the esophagus. Esophagoscopy was performed on a subsequent occasion and a nasal tube passed through the nose into the stomach for feeding purposes. On

Nov. 2, 1934, the cardia was dilated to the size of a No. 60 French sound. On November 5, dilatation to 24 ft. of water pressure was accomplished with the Plummer hydrostatic dilator. On four other occasions dilatations were carried out with a water pressure varying from 24 to 28 ft. The patient received only temporary benefit from these, so some type of operative procedure had to be carried out.

On Dec. 31, 1934, procaine hydrochloride was injected in the cervical sympathetic ganglia for diagnostic purposes. Fluoroscopic examination following the injection of the anesthetic agent revealed that the barium failed to enter the stomach. On Jan. 4, 1935, Dr. Judd operated on the patient. The abdomen was entered through a left upper rectus incision. All the tissue on the lesser curvature of the stomach near the cardia, including the left gastric artery, was ligated and dissected free in an effort to remove the sympathetic nerve supply to the cardia. Gastrotomy was performed; the hand was inserted into the stomach and the cardia was dilated so that it admitted three fingers. The obstruction was definitely due to spasm and no other pathologic change could be demonstrated. The opening in the stomach was then closed with chromic catgut sutures and one row of interrupted silk sutures.

The patient did well immediately after the operation. He was able to take fluid and food by mouth, with ease. However, his mental depression increased as he believed he had an incurable lesion. Toward the latter part of his hospital stay, he noted a gradual return of his obstructive symptoms. On February 18 and 27, dilatation from above with 26 and 24 ft. of water pressure, respectively, was carried out. This gave him relief and again he was able to take a relatively normal diet.

On March 14, 1935, the patient returned, complaining of a mass and tenderness in the right lower quadrant of his abdomen, which had been present only a few days. He had been eating normally since the last dilatation and had had no unusual pain. On March 15, through a right inguinal incision over the most prominent portion of the mass, a small appendiceal abscess was found which contained 5 c.c. of thin yellow pus. Appendectomy was performed. This additional misfortune added to the patient's mental depression, and although his convalescence from the appendectomy was not complicated, he remained dispirited and anxious. He was allowed to return home six weeks after this operation. It was hoped that the new surroundings would change his mental attitude. He seemed relatively cheerful for a time and was regaining his strength. However, during a period of depression six weeks after his discharge from the clinic, he took his own life.

CASE 6.—The patient, a carpenter, 71 years of age, registered at the clinic Jan. 5, 1938. At this time his chief complaint was dysphagia of ten years' duration. The difficulty in swallowing had become progressively worse since its onset. At the time of his admission he required two hours to ingest a meal, and he felt that only a small amount of the food and fluid passed into his stomach. The greater portion of his meals was regurgitated. The regurgitation took place chiefly at night. He had as much difficulty in taking liquid as solid food. He had lost 18 pounds (8.2 kg.) during his illness. Four years prior to his examination at the clinic, an unsuccessful attempt had been made to dilate the cardia, as he had refused to tolerate the passage of the bougies. He also complained of passing urine two to three times each night and of dysuria.

On examination the patient revealed evidence of recent loss of weight. He was dehydrated and emaciated. He was 67½ in. (170.8 cm.) tall and weighed only 127 pounds (57.6 kg.). He stated that his normal weight was 145 pounds (65.8 kg.). Bilateral indirect inguinal hernias were present, and the prostate was found to be moderately enlarged.

The laboratory findings were as follows: hemoglobin, 14.8 Gm. per 100 c.c. of blood; erythrocytes, 4,910,000 per c.mm.; and leucocytes, 7,900. The urine on

several occasions contained pus cells grade 1 or 2 on a grading basis of 1 to 4. The flocculation test for syphilis gave negative results. The roentgenologic diagnosis was cardiospasm, with marked dilatation of the esophagus, and the clinical diagnosis, cardiospasm with complete esophageal obstruction.

On Jan. 10, 1938, esophagoscopy was carried out, and 800 c.c. of thick, foul material was aspirated from the esophagus. There was considerable ulceration involving the lower esophagus. The esophagoscope could not be passed into the stomach. The esophagoscopist hesitated to force a stomach tube into the stomach for fear of perforating the badly diseased esophagus. Gastrostomy was thought more advisable for feeding purposes and at the same time a retrograde dilatation of the esophagus.

At operation on Jan. 11, 1938, in which an upper left rectus incision was used, the stomach was found to be small. There was no evidence of neoplastic change. Gastrotomy was made in the anterior surface of the stomach as near the cardia as possible, and after considerable difficulty, a finger was passed through the cardia into the esophagus. The cardia was dilated manually. After this, a sterile Rehfuss tube was introduced in a retrograde manner through the gastrotomy opening and up into the esophagus where it was pulled through by the anesthetist. A strong fishline string was then passed up the esophagus and out of the mouth. A de Pezzer catheter (No. 30), with the lower portion of the bell removed, was sutured in place as a Witzel type of gastrostomy. The opening in the stomach was closed by two rows of continuous catgut sutures and an additional interrupted third row. Omentum was placed around the tube, which was brought out through the upper portion of the incision.

The patient's immediate postoperative course was uneventful, and he was able to take fluids and food by mouth. After a few days, however, he became unable to take nourishment by mouth. Gastrostomy feedings were resorted to. He tolerated these poorly, and diarrhea developed. On Jan. 27, 1938, dilation of the cardia from above was deemed advisable. Dilatation to a No. 60 French bougie was accomplished without incident over the fishline string that had been passed at the previous operation. The patient failed to do well. He had a great deal of difficulty because of the enlarged prostate, and a retention catheter was finally necessary. Despite the intravenous administration of fluids and feedings through the gastric stoma, he became progressively weaker. On Feb. 9, 1938, bronchopneumonia developed, and he died on February 16.

CASE 7.—A priest, aged 69 years, was examined at the Mayo Clinic on July 28, 1939; his chief complaint was difficulty in swallowing. He first noticed this difficulty in 1915, when, as he expressed it, solid foods seemed to lodge beneath the lower end of his sternum. He also began to regurgitate undigested food eaten several hours previously. This tendency to regurgitate was noticed particularly at night. Anxiety and stress of any kind exaggerated the symptoms of obstruction and prohibited him from taking food for several days at a time. Early in the course of the disease the patient discovered that, by ingesting large quantities of water following his usual meal and by increasing the intrathoracic pressure, he was able to force a portion of the meal into his stomach. By this means he had maintained his normal weight and had appeared in good health until ten days prior to his admission to the clinic. In the last ten days symptoms of obstruction had become much exaggerated. He lost considerable weight, became dehydrated, and was unable to sleep because of the regurgitation of food from his esophagus. He also began to complain of rather severe pain in the epigastrium. Roentgenologic studies elsewhere in 1925, 1928, and 1929 revealed dilation of the esophagus associated with obstruction of the cardia, and a diagnosis of cardiospasm had been made.

On examination the patient was found to be weak and emaciated, having lost 26 pounds (11.8 kg.) within the preceding two weeks. He measured 65 in. (165 cm.) and weighed 124 pounds (56.2 kg.). There was definite dullness to percussion along the thoracic course of the esophagus posteriorly. Roentgenologic examination revealed the esophagus to be enormously dilated, with marked obstruction at the cardia (Fig. 7*a*). A roentgenologic diagnosis of cardiospasm was made.

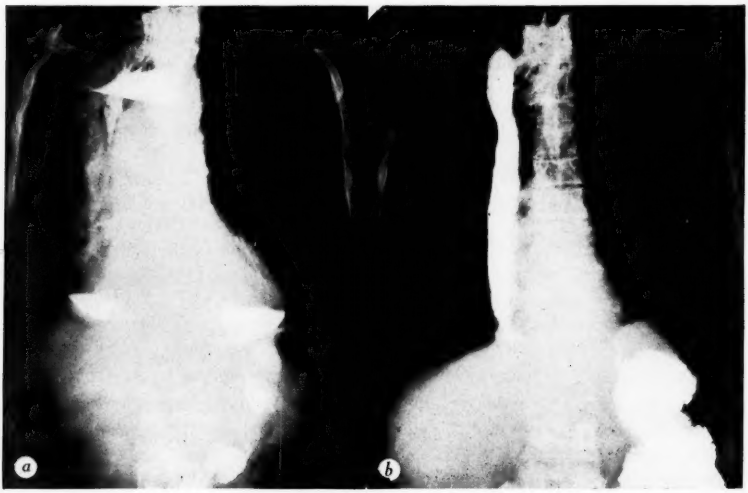

Fig. 7.—*a*, Enormous dilatation of the esophagus, with marked obstruction at the cardia. *b*, After esophagogastrostomy. The esophagus is markedly reduced in size and the opaque substance passes readily into the stomach.

The patient was hospitalized immediately and given fluids intravenously. After he had swallowed a thread and his fluid balance had been somewhat restored, dilatation was attempted. At the first attempt, dilatation to a No. 29 French sound was accomplished. The thread became disengaged so that further dilatation had to be discontinued. Subsequently, dilatation with bougies was performed four times, and with the Plummer hydrostatic dilator six times. He would obtain some relief immediately after the dilatation, but this would be only temporary. It was thought that his inability to get permanent relief was due to the exaggerated angulation of the esophagus at the cardia. On one occasion a Rehfuss tube was passed after dilatation for feeding purposes. However, the patient vomited the tube almost immediately. The situation became desperate as the patient was being maintained on intravenous fluids alone.

On Sept. 26, 1939, exposure was accomplished through a primary left upper rectus incision. The abdominal organs, as far as could be determined, were essentially normal. The intra-abdominal portion of the esophagus was more dilated than usual, measuring approximately 6 cm. in diameter. The region of the esophagogastric juncture was somewhat thickened, but otherwise essentially negative to palpation. The esophageal hiatus was split laterally for approximately 4 cm., and the distal portion of the esophagus within the mediastinum was mobilized so that a sac approximately 6 cm. in diameter was drawn within the abdominal cavity. The diaphragm around the esophageal hiatus was attached high along the esophagus to hold it within the abdomen. The distal portion of the esophagus and the anterior

portion of the fundus of the stomach were united in side-to-side anastomosis with all silk sutures; the opening between the esophagus and stomach was approximately 3 cm. in diameter.

A No. 20 French catheter was inserted into the stomach after gastrostomy in order that the stomach might be emptied in case of postoperative distention, and, also, for feeding purposes, if necessary. This was done according to the modified Witzel technique, and the catheter was brought out through a stab wound just under the left costal margin.

The patient's postoperative course was complicated by nutritional edema. This was combated by a transfusion and feedings through the tube. He was soon able to take food and fluids by mouth. Roentgenologic examination at the time of his dismissal from the hospital revealed the esophagogastrostomy to be free and the esophagus to be somewhat reduced in size (Fig. 7*b*).

In a letter written to the clinic six weeks after his dismissal, the patient stated that he had gained 40 pounds (18.1 kg.), was able to eat all types of food without difficulty, and looked and felt twenty years younger.

SUMMARY AND CONCLUSIONS

The operative treatment of cardiospasm will be found necessary in only a relatively few cases. In more than 1,200 cases of cardiospasm encountered at the Mayo Clinic, surgical procedures have been carried out in only seven instances. These cases are reported in detail. The Mikulicz operation of manual dilatation of the cardia was performed four times, esophagogastrostomy once, thoracocervical sympathectomy once, and abdominal sympathectomy combined with manual dilatation of the cardia once. In our opinion, surgical procedures at times will be found necessary in those few cases in which the esophagus is markedly dilated, tortuous, and lengthened, with angulation at the cardia and development of a reservoir below the level of the opening of the cardia.

REFERENCES

1. Bull, P. N.: So-called Idiopathic Dilatation of the Oesophagus; Synonyms: Dilatatio Fusiformis Oesophagi, Cardiospasmus, Megaoesophagus; Five Cases of Which Four Have Been Treated by Means of Oesophago-Gastrostoma Subdiaphragmatica, Ann. Surg. **81**: 59, 470, 1925.
2. Cameron, J. A. M.: Oesophagectasia in a Child, Arch. Dis. Child. **2**: 358, 1927.
3. Clairmont, P.: Quoted by Sauerbruch, F., and O'Shaughnessy, L.
4. Craig, W. McK., Moersch, H. J., and Vinson, P. P.: Treatment of Intractable Cardiospasm by Bilateral Cervicothoracic Sympathetic Ganglionectomy; Report of a Case, Proc. Staff Meet., Mayo Clin. **9**: 749, 1934.
5. Eliason, E. L., and Erb, W. H.: Cardiospasm: Report of Two Cases Treated by Resection of Sympathetic Supply to the Cardiac Sphincter, Am. J. Surg. **35**: 105, 1937.
6. Freeman, Leonard: An Operation for the Relief of Cardiospasm Associated With Dilatation and Tortuosity of the Oesophagus, Ann. Surg. **78**: 173, 1925.
7. Häggström, P.: Quoted by Sturtevant, M.
8. Hannay, A. J.: Quoted by Sturtevant, M.
9. Heller, E.: Quoted by Bull, P. N.
10. Heyrovsky, Hans: Quoted by Bull, P. N.
11. Hill, Catharine J.: A Contribution to Our Knowledge of the Enteric Plexuses. Phil. Tr. Roy. Soc., London, s. B. **215**: 355, 1927.
12. Hoffmann: Quoted by Sturtevant, M.
13. Judd, E. S., Vinson, P. P., and Greenlee, D. P.: Retrograde Dilatation of the Oesophagus for Cardiospasm, Surg., Gynec. & Obst. **48**: 494, 1929.

14. Knight, G. C.: The Relation of the Intrinsic Nerves to the Functional Activity of the Oesophagus, Brit. J. Surg. **22**: 155, 1934.
15. Knight, G. C., and Adamson, W. A. D.: Achalasia of the Cardia, Proc. Roy. Soc. Med. **28**: 891, 1935.
16. Kümmel, Hermann: Quoted by Bull, P. N.
17. Lambert, A. V. S.: Treatment of Diffuse Dilatation of Oesophagus by Operation; Description of a Hitherto Unpublished Method; Report of a Case, Surg., Gynec. & Obst. **18**: 1, 1914.
18. Lendrum, F. C.: Anatomic Features of the Cardiac Orifice of the Stomach; With Special Reference to Cardiospasm, Arch. Int. Med. **59**: 474, 1937.
19. Lindström, Erik: Quoted by Bull, P. N.
20. Meade, H. S.: Case of Sympathectomy in Treatment of Achalasia of Cardia, Irish J. M. Sc., pp. 130-131, March, 1939.
21. Meyer, Willy: Impermeable Cardiospasm Successfully Treated by Thoracotomy and Esophagoplication, J. A. M. A. **56**: 1437, 1911.
22. Moersch, H. J.: Cardiospasm; Its Diagnosis and Treatment, Ann. Surg. **98**: 232, 1933.
23. Plummer, H. S.: Cardiospasm, With a Report of Forty Cases, J. A. M. A. **51**: 549, 1908.
24. Plummer, H. S., and Vinson, P. P.: Cardiospasm; A Report of 301 Cases, M. Clin. North America **5**: 355, 1921.
25. Plummer, H. S.: Diffuse Dilatation of the Esophagus Without Anatomic Stenosis (Cardiospasm); A Report of Ninety-One Cases, J. A. M. A. **58**: 2013, 1912.
26. Plummer, H. S.: Cardiospasm, With Report of Cases, J. Minnesota M. A. **26**: 419, 1906.
27. Purton, J.: Quoted by Sturtevant, M.
28. Rake, G. W.: On the Pathology of Achalasia of the Cardia, Guy's Hosp. Rep. **77**: 141, 1927.
29. Roepke: Quoted by Bull, P. N.
30. Russel, J. C.: Diagnosis and Treatment of Spasmodic Stricture of the Oesophagus, Brit. M. J. **1**: 1450, 1898.
31. Sauerbruch, Ferdinand, and O'Shaughnessy, Laurence: Thoracic Surgery, Baltimore, 1937, William Wood & Co., 394 pp.
32. Schaldemose, V.: Quoted by Bull, P. N.
33. Sencert, L., and Simon, R.: Le Traitement operatoire de la dilatation idiopathique de l'oesophage, Rev. de chir., Paris **59**: 355, 1921.
34. Sippy, B. W.: Quoted by Sturtevant, M.
35. Sturtevant, Mills: Cardiospasm; With a Review of the Literature, Arch. Int. Med. **51**: 714, 1933.
36. Turner, G. G.: Some Experiences in the Surgery of the Oesophagus, New England J. Med. **205**: 657, 1931.
37. Mikuliez, J.: Quoted by Judd, E. S., Vinson, P. P., and Greenlee, D. P.
38. Walton, A. J.: Neuro-Muscular Obstructions of the Gastro-Intestinal Tract (Purvis oration), Lancet **2**: 1331, 1930.
39. Willis, Thomas: Quoted by Turner, G. G.
40. Womack, N. A.: Esophagoplasty for Esophageal Achalasia, S. Clin. North America **18**: 1241, 1938.
41. Zenker, in von Ziemssen: Quoted by Sturtevant, M.

DISCUSSION

DR. CARL EGGERS, New York.—In the abstract of the paper of Dr. Gray and Dr. Skinner it is stated that operative treatment is found to be necessary in relatively few cases of cardiospasm. In support of this statement, figures from the Mayo Clinic are cited which show that, of a total of more than 1,200 cases of cardiospasm, only seven were operated on.

This bears out the general belief that symptomatic relief can be obtained by dilatation, without great risk to the patient. The disadvantage of the treatment is that it has to be carried out by a specialist and that it has to be repeated at more or less frequent intervals. This means that the patient has to be transported to a clinic where such treatment may be given.

Operation is usually undertaken for only a very definite indication: (1) to bring about relief in those cases in which dilatation has failed; (2) to bring about

more permanent symptomatic relief than is usually obtained after instrumental dilatation; (3) to try to remedy the cause of the disability.

Operations such as manual dilatation of the cardia from below, or transpleural and transperitoneal esophagogastrostomy, establish better drainage of the esophagus and thereby bring symptomatic relief.

Resection of the sympathetic nerve supply to the cardia takes into consideration the etiology of the condition and tries to re-establish the delicate balance between the various elements of the nervous system concerned with the function of the cardia, and thereby acts as a curative measure.

Dr. Gray and Dr. Skinner have used all of these methods. We have operated on four cases, not because the operation was necessary, but in order to study existing conditions at the cardia and in the hope of bringing about more permanent relief than is usually obtained with instrumental dilatation.

CASE 1.—Mrs. H. C., 63 years of age, came under our care in 1931. She had developed regurgitation of saliva about ten months before. In the beginning, it contained no food particles or blood. Gradually the condition became worse. Epigastric distress developed, associated with dysphagia, regurgitation of food, and loss of weight. A diagnosis of carcinoma had been made on the basis of a roentgenologic examination. General physical examination was essentially negative. The diagnosis of carcinoma of the lower esophagus was verified at our hospital on the basis of the following roentgenologic findings: "Filling defect in the lower esophagus just above the stomach which is constant; it is obstructive to fluids; the esophagus above is moderately dilated; barium trickles through the narrowed area constantly instead of passing through in spurts, as normally; the area of involvement is about 5 cm. in length; the edges of the region of involvement show slight irregularity." Esophagoscopy showed rigidity of the tube and inability to pass the instrument into the lesion for the purpose of making a biopsy. Carcinoma was suspected.

Operation was undertaken in the hope of being able to resect the tumor and implant the esophagus into the fundus of the stomach. An epigastric incision was made just to the left of the median line and carried upward into the angle between the ensiform and the costal margin. The left lateral ligament of the liver was divided and the under surface of the diaphragm exposed. The cardia and lower end of the esophagus were completely freed out of their bed, and a tape was passed around the esophagus. The latter was soft and normal. No tumor or infiltration was felt. A large stomach tube was passed from above and guided into the stomach, without difficulty. A diagnosis of cardiospasm was made. Examination of the abdominal organs for a lesion which might reflexly be responsible for the cardiospasm was negative.

Convalescence was uneventful. The patient continued to have difficulty for a while. During the last few years she has gained weight, and by regulating her diet has succeeded in keeping herself comfortable.

CASE 2.—Mrs. C. E., 51 years of age, first began to complain of symptoms suggestive of cardiospasm early in 1934. When seen in October, 1935, she impressed us as a nervous patient. She had had two pelvic operations years before. There had been no menstruation since that time. Her chief complaints were dysphagia, salivation, and loss of 25 pounds in weight. Roentgenologic examination showed cardiospasm. She was treated medically and managed to get along until 1937, when symptoms became worse. An exploratory laparotomy was done through an incision similar to the one employed in the first case. Excellent exposure was obtained. The subdiaphragmatic portion of the esophagus was easily exposed and

mobilized. A tape was passed around it, and all connective tissue strands, blood vessels, and nerves surrounding it were divided. By gentle traction it could be palpated well up into the mediastinum where it was found to be considerably dilated. The lower portion was quite narrow. The wall of the esophagus was soft throughout, and there was no evidence of old ulceration or sphincter spasm either at the junction of the dilated and the narrowed portion, or at its entrance into the stomach. A large stomach tube was passed without difficulty.

No abnormal pathology was noted in the upper abdomen. In the pelvis there were extensive adhesions from former operations. Convalescence was uneventful.

The patient has been followed to the present time. She has gained considerable weight, and although she still has some salivation, there is no regurgitation of food and no dysphagia.

CASE 3.—Mr. L. B., 40 years of age, was referred to us in March, 1939, with an established diagnosis of cardiospasm. Symptoms had started six years previously without any known etiological factor. He had gradually become worse and had lost 20 pounds in weight. His chief complaints were dyspnea and regurgitation of food. At operation, the following findings were noted: The cardia and the lower esophagus appeared and felt normal. A tape was passed around the esophagus, and all strands and blood vessels surrounding it were doubly ligated and divided. Gentle traction permitted freeing the esophagus for from 3 to 4 inches. No gross abnormality was encountered except that the esophagus gradually became dilated in an upward direction. There was no sudden transition and no mechanical obstruction. A large stomach tube was passed into the stomach without difficulty. No abdominal lesion which could be held responsible for reflex cardiospasm was found. The convalescence was uneventful. The patient has not been completely relieved, but he is improved. He has gained some weight and regurgitation is less frequent and less severe.

CASE 4.—Mr. J. H., fifty-seven years of age, came under our observation in January, 1935, with symptoms of esophageal obstruction. He had a long history of gastrointestinal disturbance diagnosed as duodenal ulcer about 10 years before. Six years ago a gastroenterostomy had been done. Four years ago a piece of meat became lodged in the lower esophagus and had to be extracted through an esophagoscope. Some time after that he noticed disturbance at night, consisting of cough, choking sensation, and regurgitation of food. About a year ago dysphagia developed. There had been some loss of weight. Roentgenologic examination showed typical cardiospasm. Physical examination was negative. Esophagoscopy showed no ulcer or tumor. Cardiospasm was diagnosed.

He was operated upon on Feb. 9, 1935. An epigastric incision, as in the other cases, was made. A scar, evidently resulting from an ulcer, was visible and palpable on the anterior surface of the duodenum. The gastroenterostomy was patent and admitted two fingers. The cardia and the lower esophagus were freed. No abnormality was noted. A tape was passed around the esophagus for identification and traction. A one-inch incision was made in the anterior wall of the stomach between two traction sutures. By means of suction, the stomach was emptied and a finger then inserted for exploration of the cardia. A small dimple was felt, but the finger did not enter until gradual pressure had been exerted. The index finger was inserted and then the middle finger. The cardia was stretched in all directions until it admitted two fingers. Stretching with two fingers simultaneously was not very successful on account of the depth. Considerable food remnants were found in the esophagus despite the fact that it had been irrigated prior to operation. These were removed by suction. The incision in the stomach

was closed with two rows of sutures. Prior to closing the stomach, the gastroenterostomy stoma was explored from within and found to have a smooth margin, admitting two fingers.

The convalescence was uneventful. The patient has been completely relieved of all his symptoms and has gained 30 pounds in weight.

Re-examination of the esophagus in 1938 showed the condition to be the same as in 1935, with identical roentgenologic evidence of cardiospasm and no diminution in the size of the esophagus (Fig. 1).

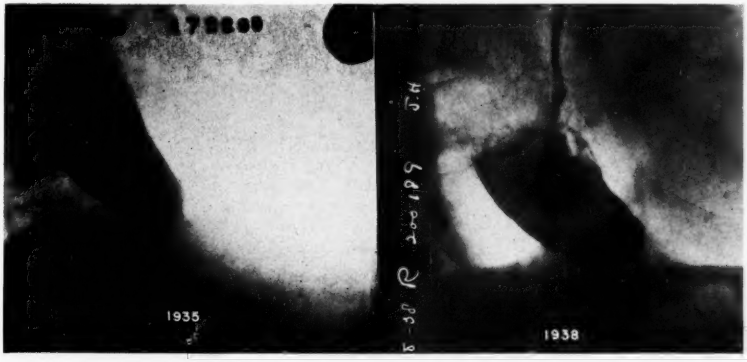

Fig. 1.—Case 4. Esophagus before operation and three years after manual dilatation. Complete relief of symptoms was effected, with a gain of 30 pounds in weight and without change in the appearance of the esophagus.

A study of this small group of cases is interesting. It shows that there is no gross anatomic change at the cardia or in the lower end of the esophagus. There is no thickening of any part of the wall similar to the lesion found in hypertrophic pyloric stenosis, and there is, therefore, no indication for an attempt at a plastic procedure.

A spasm cannot be demonstrated while the patient is under anesthesia, but fluoroscopic observations made on these patients and cinefluorographic studies have shown that there is active peristalsis in the esophagus which meets an obstruction at the cardia and sets up violent antiperistalsis. Whether this obstruction is in the nature of a spasm and is therefore entitled to be called cardiospasm, or whether it is due to achalasia of the cardia, by which is meant failure of the cardia to relax and open during the act of swallowing and which is supposed to be due to disturbance of the nervous mechanism, has not been definitely determined.

The whole question of the etiology of cardiospasm requires further study. Because the enormous dilatation of the esophagus is frequently the outstanding lesion, it has been called idiopathic dilatation and the causative factor has been considered to be a disturbance of the sympathetic nerve control similar to that found in megacolon or Hirschsprung's disease. The fact that the dilatation of the esophagus does not subside after improved drainage by stretching of the cardia seems to support this view. On the other hand, clinical and roentgenologic observations, as well as a study of the results following operations on the cardia and lower esophagus, make one feel that some interference with the function of the cardia is responsible.

The simplest operation, and one which in three of our cases led to considerable improvement, is the freeing of the cardia and lower esophagus, with division of all surrounding fibers, including nerve filaments, until the longitudinal fibers of

the esophagus can be followed down onto the wall of the stomach. It is assumed that this procedure brings about denervation of these structures.

Added to this denervation, manual stretching of the lower esophagus and cardia apparently has given far better results in one of my cases, as well as in cases reported in the literature.

If there is enormous dilatation of the esophagus with elongation and sacculation, I believe an esophagogastrostomy may be considered in selected cases.

Further studies on the nerve control of the cardia may lead to a better understanding of the etiology of cardiospasm and make it possible to bring about a cure by direct attack on the sympathetic nervous system.

At present, the state of our knowledge is still too confused and the isolated clinical and experimental observations are so much at variance that no definite conclusions can be drawn.

Rather than assume one etiology for all cases of cardiospasm, it may be well not to neglect to study the influence of neuroses or of reflex action secondary to an anatomic pathologic lesion in the abdomen, usually of the stomach and duodenum. I have a number of such patients under observation who seem to show a definite relationship between gastric and esophageal lesions.

DR. A. L. LOCKWOOD, Toronto.—I have a few films here of an enormous sigmoid cardiospasm, which I thought would be of interest to the members and guests. I could not forebear getting the sage advice of the esophagus specialists here, and of your worthy self who, I recall, has done original work on this problem.

(Film) You can see a tremendous dilatation here, and it angulates far to the right side, almost to the lateral chest wall. This is a woman 34 years of age.

(Film) This is an oblique picture. After we got the foul, retained food cleared out, two large steins of barium did not begin to fill the tremendous sacculation which she had.

(Film) We thought when we first saw this that the patient probably had a herniation through the diaphragm as well. She is 34 years of age; she had her first distress probably at 14 years of age, and was treated by dilatation here in this city (Cleveland) again when she was 17 years of age, and now this final one. It shows the cardia and the tremendous sacculation above. When she came under our care, she was sent in for an esophagogastrostomy, and it took about sixteen or eighteen days of lavage to clear out the retained food. I would not have believed that the human body could contain so much filth as she had in her esophagus. By washing it two or three times a day, it took sixteen days to get the esophagus clear so that we could even attempt to give her a thread to swallow. When one looked down the esophagoscope, it looked like one great plastic mass. We picked out specimens that were of the consistency of clay.

The curious thing about her was that she had practically no vomiting. She had a certain amount of regurgitation, but not a great deal. Her most annoying symptoms, apart from inability to swallow solid food, were nausea and distress from the odor. It was constantly with her. She was a burden to the entire family. She had lost only 7 pounds in weight.

After we got the esophagus washed out, we gave her a thread to swallow. With some little effort we got a bougie down, then a Plummer hydrostatic dilator, and dilated the esophagus to 30 pounds' pressure. She came back ten days ago and had one further dilatation; she had regained the weight.

Whether or not a cure can be promised without the use of surgery is questionable in such an extreme case, and I hope some of you who have had experience with a case of such extensive dilatation and sacculation will express an opinion. I am inclined to feel that this woman will ultimately require esophagogastrostomy. However, she is swallowing freely now and feels confident herself that she is quite cured.

DR. ROBERT JAMES, Toronto.—I have enjoyed Dr. Gray's paper very much. I thought it might be interesting to supplement what he has said with some personal experiences and some experiences of my immediate colleagues.

So far as the incidence of patients requiring operation is concerned, I fancy that many more of them should have been operated upon but were not because we feared that the operation was too dangerous. I think it is rarely necessary to do either gastrostomy or jejunostomy beforehand, although it may be wise in some very debilitated patients. I would suggest that the fluid balance and proteins may be restored by intravenous infusions and transfusion, and that before operation a duodenal tube may be passed down to the site of the obstruction to be pulled through into the stomach at operation. The patients may be fed through this tube until the edema subsides.

I think it should be called to the attention of those who have not performed an esophagogastroplasty that it is necessary to make the cut into the anterior wall of the stomach a good deal longer than one would at first think necessary, in order to remove completely the obstruction.

I am reporting this group of cases through the courtesy of my confreres. My own cases consist of three, one esophagogastrostomy, with anastomosis between the left side of the esophagus and the stomach, done just over four years ago, and two esophagogastroplasties. Dr. George Wilson has performed four anastomoses in the last four years between the left side of the esophagus and the stomach. In these patients there was no operative mortality, but one patient died some time later because of perforation of an ulcer higher in the esophagus, with a resulting mediastinal abscess.

Dr. R. I. Harris has performed one Finney type of anastomosis on the right side of the esophagus, anastomosing the lesser curvature of the stomach to the esophagus, a sigmoid type of dilatation, of course. The patient did well and was clinically relieved, but unfortunately died of lobar pneumonia some seven months later, in April, 1938.

Dr. Gordan Murray has performed one esophagogastroplasty, and Dr. Roscoe Graham one, making a total of ten cases in which there was no operative mortality. All of the living patients are clinically relieved.

Dr. Eggers called attention to another interesting point, that the x-ray findings after operation are disappointing. Actually, the patient may be clinically quite well, and yet the x-ray picture may be disappointing.

With your permission I would like to show lantern slides of my own cases. (Slide) Some of the x-rays were not very good, so I picked those that might be most useful. This is the first patient. The x-ray shows the very marked dilatation of the esophagus. The lower end was of the sigmoid type. The man was 42 years of age and had had symptoms for 7 years. He weighed 80 pounds. An anastomosis was performed between the esophagus and the stomach, an esophagogastrostomy, over four years ago. I had a note from his physician a few days ago. He is well, working steadily, swallows everything without difficulty, except chunks of meat, and weighs 120 pounds.

(Slide) That is a postoperative x-ray taken some four months after operation, and you can barely see a small amount of barium being held up. Actually, the function is very good. You can still see the dilatation of the esophagus which is not filled with barium.

(Slide) This was a patient with a very marked lesion. He had been able to swallow nothing, not even water, for some days before he came under our care. We got him into somewhat better shape by intravenous injection and transfusion and operated upon him in December, 1939, an esophagogastroplasty being done. (Slide) This picture was taken in January, 1940, and shows that function is

reasonably good. (Slide) I am afraid that does not show very well. It was taken in April, 1940, and shows that the esophagus is emptying well. The patient swallows without any recognizable difficulty and now weighs 145 pounds, as against something under 100 before operation.

(Slide) The next patient was a young man 25 years of age, who had had symptoms since 13 years of age. I think that he is perhaps the type that ordinarily should not be operated upon, but he was very much disturbed by the frequent dilatations that were necessary and was showing an adverse mental reaction. For that reason, and also for my curiosity, I am afraid, as to whether or not one could relieve these patients at an earlier stage, he was operated upon. That is the film two months after operation. He is swallowing all solid foods well. Interestingly enough, he has some holdup in drinking barium or milk.

(Slide) That is the slide of a patient before death, a man 71 years of age, admitted to the hospital with severe hemorrhage from an ulcer in the esophagus. He contracted pneumonia immediately and died, and this is the post-mortem finding. It is interesting that I had known this man for a good many years and did not know that he had anything the matter with his esophagus. Through subsequent questioning of his daughter, I found that for 15 years he had complained occasionally of indigestion and would leave the table. Nothing else was reported. Three years before, he had been kept in bed for 6 months because of supposed cardiac disease. That, of course, has been reported before, the confusion of the symptomatology of these cases with coronary disease. You see that there is an extreme dilatation of the esophagus. The little pieces of paper underneath the azygos vein which crosses the dilated esophagus at that point explain the incisura that one sees in x-rays of all these patients. The final point of interest was that we were able to study the vagi in this patient, and they showed very marked generative changes.

DR. CLARENCE BIRD, Providence, R. I.—There are three points which might be mentioned briefly that are perhaps of interest. The first is that much of the work on esophageal surgery was covered very completely in the International Surgical Congress in Madrid in 1932. We American surgeons have been very slow in taking up the work on the esophagus.

The second is that Etzel of São Paulo, Brazil, has brought forth some very convincing experimental work which tends to show that vitamin B_1 insufficiency is responsible for the initial degeneration in Auerbach's plexus.

The third point is a matter of technique, and may be explained without diagramming, I believe. This is that the Finney pyloroplasty type of procedure is to be preferred over a simple esophagogastrostomy because it does not leave a dependent spur, with a resulting sac that does not drain. You can see also that in performing a Finney pyloroplasty type of operation it is necessary for the operator to straighten the esophagus, which is very desirable. He simply cannot do this operation without pulling the esophagus way down, thus getting it in a straight line, and that, too, adds to the efficacy of the complete drainage of the esophagus.

DR. GRAY.—I should like, very sincerely, to thank Dr. Eggers, Dr. Lockwood, Dr. Janes, and Dr. Bird for their discussions.

It seems almost inconceivable, in view of the films Dr. Eggers has shown, in which there is evidence of such marked obstruction at the cardia, that there is not an actual spasm of the sphincterlike process. However, all detailed anatomic

investigation of this region has failed to show any definite circular fibers which would act as a sphincter, and for this reason it is difficult to explain the spastic process.

I am glad that Dr. Eggers has reviewed again the possible etiological factors concerned in the production of cardiospasm, for it is likely that when so many different hypotheses have been advanced, the actual cause may be attributed probably to no one single factor, but to a combination of factors.

The case which Dr. Lockwood has presented is a very interesting one. I wish that Dr. Janes or Dr. Bird might have expressed an opinion in regard to the questions Dr. Lockwood has raised, as they have had a great deal more experience than I. It would certainly seem reasonable to suppose that a patient who has such marked tortuous dilatation of the esophagus, due to cardiospasm, might expect a satisfactory result from esophagogastrostomy.

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Notices

Very shortly the Registry for Thoracic Tumors will be in operation under the auspices of the National Research Council of the Army Medical Museum. Blanks may be obtained by writing direct to Col. J. E. Ash, Curator, Army Medical Museum. As soon as possible there will also be a supply in the possession of Dr. F. R. Harper, 1008 Republic Building, Denver, Colo., and Dr. F. B. Berry, 768 Park Avenue, New York, N. Y., as well as in the office of the Secretary, Dr. Richard H. Meade, Jr., 2116 Pine Street, Philadelphia, Pa.

To begin with, the following tumors will be accepted for registration, and it is urged that the members cooperate in so far as possible in supplying the required data so that we can get off to a good start:

1. Cases of primary tumors of the lungs and bronchi, proved by the histologic examination of tissue removed at the time of operation or at autopsy. Diagnosis made by bronchoscopy or aspiration biopsy is not considered sufficiently accurate, and such cases will not be accepted by the Registry.

2. All primary neoplasms of the mediastinum, proved by histologic examination at operation or autopsy. Tumors of the lymphatic and esophageal groups are to be excepted inasmuch as the former already have their own registry and the esophagus is primarily connected with gastrointestinal pathology.

At the request of Colonel Ash, the work of the Registry at first will be limited to the above two groups until it is determined how much material will accumulate and whether or not it is feasible to expand the Registry. THE JOURNAL OF THORACIC SURGERY reserves the right to refuse to publish cases in the above groups which are not registered.

The Society of Clinical Pathologists is cooperating with us in the work of collecting and registering these tumors. We are extremely fortunate in having Dr. Tracy Mallory of Boston, Dr. N. C. Foot of New York, and Dr. John Budd, Jr., of Los Angeles as Consulting Pathologists. They do not presume to replace in any way the local pathologists, but have expressed their willingness to help the Army Medical Museum and your committee in the analysis and registration of these tumors.

The Registry at the Army Medical Museum will be open at all times for use and inspection not only by our own members, but also by members of the medical profession after permission has been obtained from our Association.

RICHARD H. MEADE, JR.
Secretary

The dates for the 1941 meeting in Toronto have not been finally decided upon, but will probably be in June. March 1, 1941, is the final day for submission of papers for the program for this meeting. Titles and abstracts should be sent to the Secretary before that date.

The Rose Lampert Graff prize of $250.00 will be awarded again this year, and the closing date for this is March 1, 1941. Papers submitted should not bear the name of the author, but should be accompanied by a sealed envelope containing the name.

Nominations for membership must be sent to the Chairman of the Membership Committee, Dr. Ethan F. Butler, Biggs Memorial Hospital, Ithaca, N. Y., before Feb. 1, 1941.

The deaths of the following members have occurred since our meeting in June:

Dr. S. Adolphus Knopf
Dr. Stuart Pritchard
Dr. Everett E. Watson

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Secretary

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EDWARDS, TUDOR---139 Harley St., W. 1, London, England
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JACKSON, CHEVALIER---3701 N. Broad St., Philadelphia, Pa.
LERCHE, WILLIAM---Cable, Wis.
LILIENTHAL, HOWARD---20 W. 77th St., New York, N. Y.
MATAS, RUDOLPH---2255 St. Charles Ave., New Orleans, La.
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ALEXANDER, JOHN---University Hospital, Ann Arbor, Mich.
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ANDRUS, WILLIAM DE WITT---New York Hospital, 525 E. 68th St., New York, N. Y.
BALLON, DAVID---1471 Crescent St., Montreal, Quebec
BALLON, HARRY C.---1414 Drummond St., Montreal, Quebec
BARNWELL, JOHN B.---University Hospital, Ann Arbor, Mich.
BECK, CLAUDE S.---Lakeside Hospital, Cleveland, Ohio
BERRY, FRANK B.---768 Park Avenue, New York, N. Y.
BETTMAN, RALPH B.---104 S. Michigan Ave., Chicago, Ill.
BIGGER, I. A.---Medical College of Virginia, Richmond, Va.
BISGARD, DEWEY---1436 Medical Arts Bldg., Omaha, Neb.
BLALOCK, ALFRED---Vanderbilt University Hospital, Nashville, Tenn.
BOHRER, JOHN V.---116 E. 58th St., New York, N. Y.
BRADSHAW, HOWARD H.---Jefferson Hospital, Philadelphia, Pa.
BUTLER, ETHAN FLAGG---Biggs Memorial Hospital, Ithaca, N. Y.
CARLSON, HERBERT A.---Northwest Clinic, Minot, N. D.
CARR, DUANE---899 Madison Ave., Memphis, Tenn.
CARTER, B. NOLAN---Cincinnati General Hospital, Cincinnati, Ohio
CHURCHILL, EDWARD D.---Massachusetts General Hospital, Boston, Mass.
COLE, DEAN B.---Professional Bldg., Richmond, Va.
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CURTIS, GEORGE M.---Ohio State University, College of Medicine, Columbus, Ohio
CUTLER, ELLIOTT C.---Peter Bent Brigham Hospital, Boston, Mass.
DAVIDSON, LOUIS R.---5 E. 53rd St., New York, N. Y.
DECKER, HARRY R.---923 Westinghouse Bldg., Pittsburgh, Pa.
DOLLEY, FRANK S.---427 S. Arden Blvd., Los Angeles, Calif.
DRASH, EVERETT C.---University of Virginia Hospital, Charlottesville, Va.
EGGERS, CARL---850 Park Ave., New York, N. Y.
ELKIN, DAN C.---618 Doctors Building, Atlanta, Ga.
ELOESSER, LEO---490 Post St., San Francisco, Calif.
FAULKNER, WILLIAM B.---384 Post St., San Francisco, Calif.
FISHER, LINCOLN---49 Carpenter Rd., Mansfield, Ohio

FLICK, JOHN B.----Apt. 3, Mermont Plaza, Merion and Montgomery Aves., Bryn Mawr, Pa.
FRANK, LOUIS WALLACE----332 W. Broadway, Louisville, Ky.
FREEDLANDER, SAMUEL O.----10515 Carnegie Ave., Cleveland, Ohio
GALE, JOSEPH W.----1300 University Ave., Madison, Wis.
GIBBON, JOHN H., JR.----4035 Pine St., Philadelphia, Pa.
GRAHAM, EVARTS A.----Barnes Hospital, St. Louis, Mo.
GRAY, HOWARD K.----102 Second Ave., S. W., Rochester, Minn.
GURD, FRASER B.----Medical Arts Bldg., Montreal, Quebec
HAIGHT, CAMERON----University Hospital, Ann Arbor, Mich.
HARPER, FREDERICK R.----1008 Republic Bldg., Denver, Colo.
HARRINGTON, STUART W.----Mayo Clinic, Rochester, Minn.
HART, DERYL----Duke Hospital, Durham, N. C.
HAYES, JOHN N.----Rockdrift, Glenwood Estates, Saranac, N. Y.
HEAD, JEROME R.----55 E. Washington St., Chicago, Ill.
HEINBECKER, PETER----Washington University Medical School, St. Louis, Mo.
HERBERT, WILLIAM P.----Flat Iron Bldg., Asheville, N. C.
HOLMAN, EMILE----Stanford University, San Francisco, Calif.
HUDSON, W. A.----602 David Whitney Bldg., Detroit, Mich.
JACKSON, CHEVALIER L.----The Medical Tower, 255 S. 17th St., Philadelphia, Pa.
JANES, ROBERT M.----170 St. George St., Toronto, Ontario
JOANNIDES, MINAS----3810 Broadway, Chicago, Ill.
JOHNS, FRANK S.----Johnston-Willis Hospital, Richmond, Va.
JONES, JOHN C.----427 S. Arden Blvd., Los Angeles, Calif.
KING, DONALD S.----1101 Beacon St., Brookline, Mass.
KINSELLA, THOMAS J.--1251 Medical Arts Bldg., Nicollet at 9th, Minneapolis, Minn.
LAMBERT, ADRIAN V. S.----768 Park Ave., New York, N. Y.
LEAHY, L. J.----105 Medical Arts Bldg., 40 North St., Buffalo, N. Y.
LEE, WALTER ESTELL----1833 Pine St., Philadelphia, Pa.
LINDSKOG, GUSTAF E.----New Haven Hospital, New Haven, Conn.
LOCKWOOD, A. L.----300 Bloor St., East, Toronto, Ontario
MCINTOSH, CLARENCE A.----1390 Sherbrooke St., W., Montreal, Quebec
MEADE, RICHARD H., JR.----2116 Pine St., Philadelphia, Pa.
MOERSCH, HERMAN----Mayo Clinic, Rochester, Minn.
MOORE, JULIAN A.----6 Battery Park Ave., Asheville, N. C.
MOORE, RICHMOND L.----180 Fort Washington Ave., New York, N. Y.
MYERS, J. ARTHUR----730 LaSalle Bldg., Minneapolis, Minn.
NEUHOF, HAROLD----4 E. 95th St., New York, N. Y.
NEWTON, HARLAN F.----319 Longwood Ave., Boston, Mass.
NIXON, J. W.----1121 Nix Professional Bldg., San Antonio, Texas
O'BRIEN, EDWARD J.----10 Peterboro St., Detroit, Mich.
OCHSNER, ALTON----Tulane University, New Orleans, La.
ORNSTEIN, GEORGE----115 Central Park West, New York, N. Y.
OVERHOLT, RICHARD H.----1101 Beacon St., Brookline, Mass.
PACKARD, EDWARD N.----105 Main St., Saranac Lake, N. Y.
PETERS, LEROY S.----221 W. Central Ave., Albuquerque, N. M.
PICKHARDT, OTTO C.----117 E. 80th St., New York, N. Y.
PINNER, MAX----Montefiore Hospital, Gun Hill Road, New York, N. Y.
PROCTOR, OSCAR S.----Medical-Dental Bldg., Seattle, Wash.
RIENHOFF, WILLIAM F., JR.----1201 N. Calvert St., Baltimore, Md.
RIGLER, LEO G.----University Hospital, Minneapolis, Minn.
ROGERS, W. L.----490 Post St., San Francisco, Calif.
SAMSON, PAUL C.----411 30th St., Oakland, Calif.

SINGER, J. J.—2200 W. Third St., Los Angeles, Calif.
SINGLETON, ALBERT O.—320 21st St., Galveston, Texas
SMITH, DAVID T.—Duke University, Durham, N. C.
STEINKE, CARL R.—159 S. Main St., Akron, Ohio
STEPHENS, H. BRODIE—384 Post St., San Francisco, Calif.
STRIEDER, JOHN W.—171 Bay State Road, Boston, Mass.
THEARLE, WILLIAM H.—221 W. Central Ave., Albuquerque, N. M.
THORBURN, GRANT—105 E. 53rd St., New York, N. Y.
TOUROFF, ARTHUR S. W.—940 Park Ave., New York, N. Y.
TROUT, HUGH—Jefferson Hospital, Roanoke, Va.
TRUESDALE, PHILEMON E.—151 Rock St., Fall River, Mass.
TUCKER, GABRIEL—250 S. 18th St., Philadelphia, Pa.
TUTTLE, WILLIAM M.—305 Professional Bldg., 10 Peterboro St., Detroit, Mich.
URQUHART, ROBERT GLEN—Uncas-on-Thames, Norwich, Conn.
VAN ALLEN, CHESTER M.—Sir Dorabji Tata Trust, Bombay House, Fort Bombay, India
VAN HAZEL, WILLARD—30 N. Michigan Ave., Chicago, Ill.
WANGENSTEEN, OWEN H.—University Hospital, Minneapolis, Minn.
WEINBERG, JOSEPH A.—1012 Medical Arts Bldg., Omaha, Neb.
WELLES, EDWARD S.—110 Park Ave., Saranac Lake, N. Y.
WOODRUFF, WARRINER—8 Church St., Saranac Lake, N. Y.

Associate Members

ADA, ALEXANDER E. W.—120 E. 75th St., New York, N. Y.
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AYCOCK, THOMAS B.—101 W. Read St., Baltimore, Md.
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BERCK, MAURICE—933 5th Ave., New York, N. Y.
BETTS, REEVE HAWKINS—1101 Beacon St., Brookline, Mass.
BIRD, CLARENCE—116 Waterman St., Providence, R. I.
BLADES, BRIAN—Barnes Hospital, St. Louis, Mo.
BLOCH, ROBERT G.—University of Chicago, Chicago, Ill.
BORTONE, FRANK—2765 Hudson Blvd., Jersey City, N. J.
BREIDENBACH, WARREN G.—970 Fidelity Bldg., Dayton, Ohio
BROWN, A. LINCOLN—490 Post St., San Francisco, Calif.
BUCKINGHAM, WILLIAM W.—800 Professional Bldg., Kansas City, Mo.
BURNETT, W. E.—3401 N. Broad St., Philadelphia, Pa.
CABITT, HENRY L.—1248 Beacon St., Brookline, Mass.
CHAMBERLAIN, JOHN MAXWELL—Homer Folks Hospital, Oneonta, N. Y.
COOPER, DAVID A.—1520 Spruce St., Philadelphia, Pa.
COX, WILLIAM V.—384 Main St., Lewiston, Me.
DAVIS, EDGAR W.—1150 Connecticut Ave., Washington, D. C.
DAVISON, RICHARD—4022 Sheridan Road, Chicago, Ill.
DAY, J. CLAUDE—10 Peterboro St., Detroit, Mich.
DE BAKEY, MICHAEL E.—1430 Tulane Ave., New Orleans, La.
DELANEY, WILLIAM E., JR.—416 Pine St., Williamsport, Pa.
DESHAIES, GEORGES—3300 Boulevard Govine Ouest, Montreal, Quebec
DIEFFENBACH, RICHARD H.—570 Mount Prospect Ave., Newark, N. J.
DOUGLASS, RICHMOND—Biggs Memorial Hospital, Ithaca, N. Y.
EGLEE, EDWARD P.—105 E. 53rd St., New York, N. Y.
EHLER, ADRIAN E.—Albany Hospital, Albany, N. Y.
FISHBACK, FREDERICK C.—1801 Eye St., N. W., Washington, D. C.

FINNEY, GEORGE G.—2947 St. Paul St., Baltimore, Md.
FORSEE, JAMES H.—Walter Reed General Hospital, Washington, D. C.
GARLOCK, JOHN H.—50 E. 77th St., New York, N. Y.
GEBAUER, PAUL—10515 Carnegie Ave., Cleveland, Ohio
GOLDMAN, ALFRED—516 Sutter St., San Francisco, California
GRACE, EDWIN J.—121 Fort Greene Place, Brooklyn, N. Y.
GROSS, ROBERT E.—300 Longwood Ave., Boston, Mass.
GUPTELL, PLIMPTON—16 N. Goodman St., Rochester, N. Y.
HARTER, JOHN S.—Sanatorium, Miss.
HERBEN, GEORGE F.—Twitchell Lake, Big Moose, N. Y.
HUN, HENRY—149 Washington Ave., Albany, N. Y.
JANES, ERNEST C.—250 Main St., E., Hamilton, Ontario
JOHNSON, HOLLIS E.—2122 West End Avenue, Nashville, Tenn.
KERGIN, FREDERICK—170 St. George St., Toronto, Ontario
KNOEPP, LOUIS F.—5900 Block Line Ave., Shreveport, La.
LAIRD, ROBERT—170 St. George St., Toronto 5, Ontario
LEETCH, HENRY W.—108 Main St., Saranac Lake, N. Y.
LESTER, CHARLES W.—70 E. 80th St., New York, N. Y.
LEVEN, N. LOGAN—1360 Lowry Medical Arts Bldg., St. Paul, Minn.
LONGACRE, JACOB JAMES—Cincinnati General Hospital, Cincinnati, Ohio
MCPHAIL, ROSS—Mountain View Sanatorium, Lakeview, Wash.
MACDONALD, R. H.—401-2 Canada Bldg., Saskatoon, Saskatchewan
MAIER, HERBERT C.—140 E. 54th St., New York, N. Y.
MAUTZ, F. R.—University Hospital, Cleveland, Ohio
MAYER, EDGAR—470 Park Ave., New York, N. Y.
MELTZER, HERBERT—Ninette, Manitoba
MERKEL, CARL G.—Mount Morris Tuberculosis Hospital, Mount Morris, N. Y.
MEYER, HERBERT WILLY—170 E. 78th St., New York, N. Y.
MIDELFART, PETER A.—314 Grand Ave., East, Eau Claire, Wis.
MILLER, FELIX P.—109 N. Oregon St., El Paso, Texas
MUDD, JAMES L.—634 N. Grand Blvd., St. Louis, Mo.
MURRAY, FOSTER—80 Hanson Place, Brooklyn, N. Y.
NEHIL, LAWRENCE W.—707 Heyburn Bldg., Louisville, Ky.
OATWAY, WILLIAM H., JR.—2004 University Ave., Madison, Wis.
POLLOCK, WILLIAM C., LIEUT. COL. M. C., U. S. A.—Letterman General Hospital, San Francisco, Calif.
POTTER, BENJAMIN P.—100 Clifton Place, Jersey City, N. J.
POWERS, JOHN H.—Mary Imogene Bassett Hospital, Cooperstown, N. Y.
RAINE, FORRESTER—425 E. Wisconsin Ave., Milwaukee, Wis.
RANDOLPH, V. S.—15 E. Monroe St., Phoenix, Ariz.
RIGGINS, H. MCLEOD—140 E. 54th St., New York, N. Y.
RINGER, PAUL H.—Arcade Bldg., Asheville, N. C.
SCHAFFNER, VERNON D.—Kentville, Nova Scotia
SHAW, ROBERT R.—723 Medical Arts Bldg., Dallas, Texas
SHIPMAN, SIDNEY—490 Post St., San Francisco, Calif.
SIMPSON, H. MURRAY—327 Queen's Ave., London, Ontario
SKINNER, A. M.—Homer Folks Tuberculosis Hospital, Oneonta, N. Y.
SKINNER, GEORGE F.—36 Coburg St., St. John, New Brunswick
SOMMER, GEORGE N. J., JR.—120 W. State St., Trenton, N. J.
STEELE, J. D.—324 E. Wisconsin Ave., Milwaukee, Wis.
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TYSON, M. DAWSON—Hanover, N. H.

VIDAL, J. A.----454 Sherbrooke St., E., Montreal, Quebec
VINEBERG, ARTHUR M.----1414 Drummond St., Montreal, Quebec
VORWALD, ARTHUR----Saranac Laboratory, Saranac Lake, N. Y.
WILLIAMS, MARK H.----110 Murray St., Binghampton, N. Y.

Senior Members

ALLEN, DUFF S.----Washington University Medical School, St. Louis, Mo.
AUCHINCLOSS, HUGH----Presbyterian Hospital Medical Center, New York, N. Y.
BAZIN, A. T.----1414 Drummond St., Montreal, Quebec
BOLAND, FRANK K.----478 Peachtree St., N. E., Atlanta, Ga.
BRANOWER, WILLIAM----285 Central Park West, New York, N. Y.
BRUNN, HAROLD----384 Post St., San Francisco, Calif.
BYERS, J. RODDICK----3166 Westmount Blvd., Montreal, Quebec
CLERF, LOUIS H.----1530 Locust St., Philadelphia, Pa.
CROWE, SAMUEL J.----Johns Hopkins Hospital, Baltimore, Md.
DAVISON, T. C.----478 Peachtree St., N. E., Atlanta, Ga.
DIEDERICH, VICTOR----236 Central Ave., Hot Springs, Ark.
DOVELL, CHAUNCEY D., MAJOR, M. C., U. S. A.---Station Hospital, Fort Sheridan, Ill.
DUNHAM, H. KENNON----27th Floor, Union Central Life Bldg., Cincinnati, Ohio
EINHORN, MAX----20 E. 63rd St., New York, N. Y.
FERGUSON, R. G.----Fort San, Saskatchewan
FISCHER, HERMAN----35 E. 84th St., New York, N. Y.
HARVEY, SAMUEL----New Haven Hospital, New Haven, Conn.
HEUER, GEORGE J.----Cornell University Medical College, 1300 York Ave., New York, N. Y.
KELLER, WILLIAM L., COL. M. C., U. S. A.---Walter Reed Hospital, Washington, D. C.
KERNAN, JOHN D.----103 E. 78th St., New York, N. Y.
LEMON, WILLIS S.----510 10th Ave., S. W., Rochester, Minn.
LEWALD, LEON T.----1200 5th Ave., New York, N. Y.
LORD, FREDERICK T.----305 Beacon St., Boston, Mass.
MCSWEENEY, E. S.----102 E. 35th St., New York, N. Y.
MAES, URBAN----1671 Octavia St., New Orleans, La.
MANGES, MORRIS----1185 Park Ave., New York, N. Y.
MATSON, RALPH C.----1004 Stevens Bldg., Portland, Ore.
MILLER, JAMES A.----133 E. 64th St., New York, N. Y.
MILLER, ROBERT, JR.----Mountain Lake, Lake Wales, Fla.
MULLER, GEORGE P.----1930 Spruce St., Philadelphia, Pa.
PETERSON, EDWARD W.----525 Park Ave., New York, N. Y.
POOL, EUGENE H.----107 E. 60th St., New York, N. Y.
ROBINSON, SAMUEL----22 W. Micheltorena St., Santa Barbara, Calif.
SHIPLEY, ARTHUR M.----University Hospital, Baltimore, Md.
STETTEN, DEWITT----850 Park Ave., New York, N. Y.
STEWART, GEORGE A.----3301 North Charles St., Baltimore, Md.
STEWART, WILLIAM H.----1010 5th Avenue, New York, N. Y.
WEBB, GERALD B.----402 Burns Bldg., Colorado Springs, Colo.
WHIPPLE, ALLEN O.----180 Fort Washington Ave., New York, N. Y.
WHITTEMORE, WYMAN----199 Beacon St., Boston, Mass.
WILENSKY, ABRAHAM O.----12 E. 87th St., New York, N. Y.